THE BRITISH JOURNAL
OF
TUBERCULOSIS

Vol. XXX. April, 1936. No. 2.

EDITORIAL

At a meeting of the Tuberculosis Association, Dr. Bardswell remarked that one of the advantages of artificial pneumothorax treatment was that it necessitated the regular attendance of the patient at the clinic, and this kept him under medical supervision. This is very true and applies to other methods such as gold or tuberculin, and in trying to estimate the value of any treatment one should not overlook the part played by constant medical supervision.

How often will a patient regard a phrenic operation or the fact of being in Switzerland as a cure, and will fail to see the need for medical advice. It may be said, " Give me a good medical superintendent, and I do not care where you build your sanatorium."

Drs. Hudson and Wollaston claim good results in certain cases of pneumothorax so partial that many would regard it as useless. They state, however, that such treatment should not be maintained except by those with experience, and here again we get the added factor of skilled advice, quite apart from the actual pneumothorax.

The days are past when it was difficult to persuade a patient or even his medical adviser to agree to pneumothorax, except in the most urgent case. The position of artificial pneumothorax in the treatment of pulmonary tuberculosis is established, but opinion is becoming less in favour of regarding this as the sole or even principal method of treatment. It is but one method of giving additional rest to the lung.

There are many who regard a natural cure as the best, and, whilst admitting its value, feel that artificial pneumothorax should not lightly be undertaken.

If unsuccessful, surgical methods often become necessary. It is stated that the cauterisation of adhesions is not a serious matter, and will often convert a useless into a useful pneumothorax. And yet it is by no means uncommon to see this procedure followed by an effusion which may become purulent or lead to gradual obliteration of the pleural cavity and necessitate thoracoplasty in one, two or more stages.

To push someone into a river and then plunge in and save him may be spectacular, but why push him in? Thoracoplasty has saved many lives, but when confronted with a patient to whom one has to advise this operation it is often impossible to refrain from thinking how much better it would have been had simple treatment been directed to prevent him from getting into such a state from which only thoracoplasty could save him.

The tuberculosis death-rate has been steadily falling for many years, but it is impossible to note in the graph any special fall, and to say this was due to pneumothorax, that to thoracoplasty. The only real interference with the graph is an increase during the years of the war.

It would be a calamity if opinion were to return to that of twenty years ago, when pneumothorax was regarded as a dangerous experiment, but we welcome the growing tendency to appreciate the value of simple rest and medical treatment, and to regard collapse therapy as an additional method —a great help in time of need, but often unnecessary.

PROBLEMS IN APPLIED MEDICINE

WHEN SHOULD REFILLS BE STOPPED ?*

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One of the most difficult problems encountered in artificial pneumothorax treatment of pulmonary tuberculosis is when to permit complete re-expansion of the lung. The main purpose of this paper is to discuss the points which the author considers important factors in his decision, and to describe the technique that he favours during the process of re-expansion. Only such cases will be considered as those in which a satisfactory degree of collapse has been obtained and maintained, and in which, if contralateral disease has been present, the patient has become symptom-free.

Most of the early pneumothorax cases were not submitted to the treatment long enough, and subsequently there were numerous relapses. As time went on, however, and this form of therapy became more widespread, its term gradually increased. From six months to one year of collapse was not an uncommon period fifteen years ago, while now one seldom sees a case where the total period of satisfactory pneumothorax is less than three years. Rist,[1] writing in 1930, stated that he had come to the conclusion that complete re-expansion should not be allowed until during the fifth year. The author[2] stated several years ago that he " aims to make his minimum period of treatment at least three years after the last positive sputum, but if there is still existing excavation roentgenographically he prefers to have it obliterated before the three-year period commences."

In the United States of America during the past five years there has been an enormous increase in the number of cases given pneumothorax. Fifteen years ago a considerable number of patients had to have complete treatment administered in sanatoria because there were no pneumothorax doctors within several hours' journey of their homes. This circumstance accounted for the fact that many patients had the treatment for periods of

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not more than six months to one year. To-day, with many doctors in the United States equipped to give pneumothorax treatments, it is exceptional when one has to travel for more than an hour for refills after his return home from a sanatorium.

The Council on Medical Education and Hospitals[3] of the American Medical Association reported that in 1934 there were 66,861 patients with pulmonary tuberculosis admitted to 494 institutions. The same report states that 16,401, or 24·5 per cent., of these patients received initial pneumothorax treatment during the same year. It seems probable to this writer that the high peak in the number of pneumothorax cases in the United States of America will be reached in another five years. As has been the experience of other civilised countries during the present century, the United States has seen a continuous and marked fall in the mortality and morbidity rates of pulmonary tuberculosis. The death-rate per 100,000 population in New York State has fallen from 124·0 in 1919 to 51·8 in 1934.

To-day one comes across far too many patients whose disease has recurred after the completion of pneumothorax treatment. These relapses are much more dangerous than was the original lesion before treatment commenced, because only a very small percentage of cases can get effective pneumothorax in a succeeding attempt, due to the new pleural adhesions formed after the lung re-expansion. Peters[4] reported that of 51 patients having a satisfactory and effective collapse optionally discontinued before two years of treatment (except 5 extending to three and four years), 32 per cent. relapsed and 12 per cent. died within a few years. All pneumothorax workers must find that the longer the treatment has lasted the fewer are the relapses after re-expansion. The literature bears ample testimony to this point. Dufault and Laroche[5] reported 65 cases of clinically effective pneumothorax. All of the patients had demonstrable cavities and positive sputum at the beginning of the treatment, but at the end they were afebrile and had negative sputa. All but 11 had been re-expanded within the previous five years. In the 52 patients who had had pneumothorax for less than three years the cure was only maintained in 46·2 per cent., whereas in the 13 patients who had had treatment for more than three years the cure was maintained in 84·6 per cent.

In carrying out pneumothorax treatment, the author attempts to follow a definite routine. After the degree of collapse has been obtained that is required to obliterate all excavation and that is considered necessary for a satisfactory splinting of the whole lesion, and as soon as symptoms of activity have disappeared, a very slow and gradual re-expansion of the lung is allowed. If one is dealing with a case having infiltration and excavation

in one upper third, and if an 80 per cent. collapse must be made of this upper third before it is satisfactory, it may be found necessary to maintain this degree of collapse for a period of one year. At the end of the second year a gradual re-expansion may find a collapse of 50 per cent. It is during the latter part of the third year and during the fourth year, when re-expansion of the same upper third now finds only a collapse of 10 to 20 per cent. before refill, that special care is necessary. One has now approached the final hurdle. It is while the treatment is at this stage that several factors have to be weighed.

Extent of Original Lesion.

The extent and nature of the original lesion is probably the most important factor in making our decision as to when we should stop treatment. The smaller the original lesion, the more confidence one can feel regarding expansion. Where there has been a large area of excavation originally. one usually finds that a considerable period has been taken up on obtaining a complete collapse, and a further long period has followed before the sputum has remained negative for tubercle bacilli. A period of three years with negative sputum has now probably brought the treatment into the fifth year. Cases like this may have had so much original lung destruction that a phrenicectomy or one or more stages of thoracoplasty may be necessary to complete the obliteration of the pleural cavity. The writer feels that the cases in this group which have continued to have intermittent positive sputa ought to have their treatment continued indefinitely. Some of these cases may have been three years negative, but still show little, if any, signs of re-expansion. If these latter cases refuse the surgery necessary to close the pleural space, it might be a supererogative to continue refills except at very long intervals—that is, if the mediastinum is fixed. However, if the mediastinum is not fixed, a shorter refill interval should be necessary to keep it in position.

X-Rays during Re-expansion.

A complete X-ray serial of each case should be in the hands of the current operator. When the patient leaves a sanatorium, or changes doctors, all of his X-ray films should be transferred with him. A tuberculosis lesion undergoes such radical changes during pneumothorax treatment that a record of these changes, by means of an X-ray serial, is indicated. To emphasise this point, it is no exaggeration to say that if the average pneumothorax patient who originally had an advanced lesion and who had the treatment completed after four years were to have had X-rays taken at three-month intervals during the course of treatment, making a total of sixteen films, each film would have been different in some respect.

Either there would be a difference in the degree of collapse, or there would be a difference in the appearance of the lesion, or both.

The most important change to look for roentgenographically in lung re-expansion is a reappearance of excavation, for such an occurrence warrants an immediate reversal of treatment, with closure of the cavity. Thickening of the pleura often obscures a considerable amount of lung detail. Usually, a radiograph of a 90 per cent. re-expanded lung in the fourth year of treatment reveals numerous small calcified deposits with several striations and bands where originally the lesion was located. One must view with alarm lesions which are shown by an X-ray serial to have undergone little resolution, to contain little or no calcium, and to have made little or no attempt at stellar formation or fibrosis, and yet have no perifocal exudate. Such innocent-looking lesions, if large, undoubtedly contain tubercle bacilli. If these lesions have not healed under pneumothorax, they certainly will not do so after re-expansion, and they may go on to central caseation, followed by liquefaction and excavation, if collapse is not maintained.

Fluid as a Complication.

The development of a serous effusion toward the end of artificial pneumothorax treatment is much less common than during the early part of the treatment. When this complication does occur, a determined effort must be made immediately to prevent it from interfering with the desired degree of collapse. By performing frequent aspirations and providing the necessary air replacements, this untoward result may be averted.

Age of Patient.

The younger the patient, the more inclined is the author to prolong pneumothorax treatment. He has not yet resorted to re-expansion in the case of a patient in his teens, and feels that one rarely is justified in so doing. Between the ages of fifteen and thirty years is the time of life when pulmonary tuberculosis is most prevalent, and it is also the period in which the patient is most active. Statistics show that relapses following re-expansion are more common during this period than later.

Occupation of Patient.

It has been the custom of the writer to encourage pneumothorax patients who follow occupations involving considerable physical exertion to change to easier jobs. One who plies a pen has a much better chance of living to an old age than one who wields a sledge-hammer or follows a plough. As most pneumothorax patients are young, such a change in their work is not a great trial, especially as their sanatorium stay has already taken

them away from their previous occupations. Sometimes circumstances prevent patients from following such a course, and there are individuals who are too set in their ways to change. The unfortunate number who have to return to hard manual labour ought to have pneumothorax treatment continued considerably longer than other patients, and a larger proportion of this group than of any other should have pneumothorax continued indefinitely.

Economic Status of Patient.

The clinic or poor patient is undoubtedly less able to develop a resistance to tuberculosis or to heal quickly his disease than is his more fortunate brother who has means. The unsanitary surroundings, poor food, and lack of fresh air, so common in the tenement districts of our large cities, are unfavourable to the rapid healing of tuberculosis lesions. It is well to remember also that similar conditions can exist even in the smaller or factory and industrial towns. Patients coming under this category ought to have their pneumothorax continued longer than the average.

Epidemiological Aspect.

The more contacts there are to any particular pneumothorax case, the more careful one has to be about terminating the treatment. A mother in the lower class, with a large family of children, ought to have her pneumothorax continued longer than otherwise, because of the havoc that may be caused if relapse occurs. The utmost care ought to be taken of such patients during re-expansion, with one's efforts directed toward finding any suspicious signs of reactivity.

Pregnancy and Re-expansion.

A considerable number of relapses in women after re-expansion probably occur because of pregnancy and the care of the child. The writer is often asked if he feels that it is safe for a certain pneumothorax patient to have a child. Where there has been a great deal of original lung destruction, pregnancy is not advised. Where the involvement has been somewhat less, and the economic and home conditions are satisfactory, pregnancy may be considered safe, provided pneumothorax is continued for at least one year after full term. This applies to patients with whom pneumothorax has been satisfactory and who have had a negative sputum for three years. In cases where the original lesion was moderate in extent, the patient is advised to choose between having the baby during the fourth year of pneumothorax treatment, following a satisfactory collapse and negative sputum, and continuing the treatment for another year, or allowing re-expansion during the fourth year and then waiting for another three years before becoming pregnant.

A Final Period of Trial.

When it has been decided that the time for stopping the treatment has arrived, the following procedure is adhered to : The partially re-expanded lung is allowed to re-expand further, so that there is only a collapse of about 10 per cent. before refill at the deepest part of the pneumothorax. The collapse is maintained at this level for from four to six months. It may be found necessary during these months to shorten the intervals between and to give smaller amounts of air, in order to insure a collapse of not more than 20 per cent. after refill. Thus, the site of the original lesion is in a largely re-expanded state all through the refill cycle. During this period the patient is instructed to give the lung a real trial by rehearsing any extra work, or exercise, that is contemplated for the following five years. The patient ought at least to be leading what is considered, for him, a normal life. If there has been no change roentgenographically following this period, and if the sputum has remained negative for tubercle bacilli, the refills are stopped and the pleural space is allowed to obliterate.

The Final Sputum Test.

Recently there has been completed an analysis of the results of sputum examinations in the pulmonary clinic of the New York Hospital and Cornell University Medical College. Out of 474 specimens of sputum examined by direct smear, approximately 15 per cent. were positive for acid-fast bacilli. Three hundred and twenty-eight cases were submitted to a three-day concentrated sputum examination, with approximately 8 per cent. more positive cases. Of 198 negative concentration examinations, an additional 10 per cent. were positive by guinea-pig inoculation. In view of these findings, it was decided to drop the direct smear examinations from the routine search for tubercle bacilli. Woolley[6] states that "even with concentration, there may be bacilli present which we do not detect, for at least 5,000 bacilli per cubic centimetre of material must be present in any smear before bacilli can be readily seen with the microscope." He presented a table in which was shown how many tubercle bacilli would appear in a certain number of fields if the carefully counted tubercle bacilli had been mixed in varying amounts of bronchiectatic sputum and stained smears made in the usual way. One example in the table was that if 10,000 bacilli were mixed in 1 c.c. of sputum, only an average of two bacilli would be found in 250 fields. From the above statistics it can readily be seen why the guinea-pig inoculation test is far superior to these other methods. Although it is an expensive procedure, it is felt that it ought to be the final test for activity before refills are stopped. Two months before the last

refill is due a three-day specimen of sputum should be used for guinea-pig inoculation. In this way the result can be obtained just before the last refill. If the guinea-pig is negative the treatment is stopped, but if the pig becomes tuberculous it is absolute proof that somewhere the disease is active, and therefore the treatment is continued for another period, except when one is fairly certain that the bacilli have come from a small pre-existing lesion in the contralateral side. The writer cites a striking example of the efficacy of this guinea-pig test. In September, 1932, just as the refills were about to be stopped, a patient was found to have positive sputum by the guinea-pig test. Roentgenographically, the original cavity had not re-opened and most of the scattered infiltration had become absorbed. However, there was a circular area of infiltration measuring about 2 by 2 cm. in the second space anteriorly of the largely re-exposed lung. Serial X-rays had shown this area in about the same condition during the three years of pneumothorax treatment. It was decided to continue refills sufficient to keep the lung in a largely re-expanded state. The patient continued to work, and in January, 1933, there was no change in the lesion evident in the new film. Three months later she suddenly began to expectorate, and direct smears revealed a high Gaffky count of tubercle bacilli. A radiograph was then taken, and it showed that the circular area mentioned above had been completely excavated. It was an easy matter to collapse the new cavity, and now, in the early part of 1936, the patient is again being re-expanded. It is the opinion of the writer that every case ought to have the benefit of this final guinea-pig concentrated sputum test. To be sure, housing of healthy guinea-pigs is an expensive item, but it is felt that the community would be repaid many times over by equipping the laboratory of each pneumothorax clinic with the facilities for this test. Even if by this means only a very small percentage of cases should be proved to be positive, the resultant continuation of pneumothorax treatment would be a valuable safeguard to patients and their contacts.

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GENERAL ARTICLES

TUBERCULOSIS IN EGYPT

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In response to a request from the Editor of this journal to report on Tuberculosis in Egypt, I propose to discuss the matter under three aspects : the incidence of the disease in this country ; the combative measures which are being taken by the authorities ; and finally, the question (which may be of a more practical interest to some readers of this journal) as to how far European patients should be encouraged to visit Egypt for the benefit of their health.

A. The Frequency of Consumption in Egypt.

We have proofs that tuberculosis has been endemic to Egypt for thousands of years. Mummies showing evidence of Pott's disease and of tuberculous coxitis have been found belonging to the Old, the Middle and the New Kingdom (Sir Armand Ruffer), covering thus the third and second millennium before Christ. In the early Middle Ages there is a remark of the Persian physician Ibn Sina (Avicenna of Bukhara, 980-1037) on the contagious nature of phthisis, proving by this observation that the disease was known to him as distinct from other troubles of the chest. If it was known in Persia at that time its coexistence in Egypt seems a safe assumption. In the eighteenth century tuberculosis was but rarely observed in Egypt : a point which will be discussed later on. In our times, again, it is very common, exacting a heavy toll of both the urban and the rural population.

Older statistical figures for the period between 1901 and 1909 have been compiled by Levy, but I am not informed as to the reliability of death registration in those years. Since 1918 official statistics are being issued by the Statistical Department of the Ministry of Finance.

The population of Egypt may be estimated at 16·4 millions at the end of 1935 ; it increases by a third of a million every year. The towns with

a limited health survey show the following population : Cairo, 1,270,000 ; Alexandria, 681,000 ; Port Said, 118,000 ; Tanta, 102,000. Added to these the population of some smaller towns and rural districts amounting to about 2·3 millions results in a total of 4·5 million people who are within reach of medical officers under the public health authorities. All statistical figures comprise only this minority of 35 per cent. of the population of Egypt. As the social, hygienic and climatic conditions under which the remaining 65 per cent. live often differ considerably from those of the first fraction it becomes obviously impossible to draw conclusions concerning the whole population from the official figures covering that fraction of 35 per cent. only.

As for that fraction : What confidence can be reposed in the statistical data ? In arranging the latest official figures of the fourth quarter of 1934 the following information is obtained : in Cairo, for example, there were 215 people reported to have died of pulmonary tuberculosis during those three months, 162 of bronchitis, and 921 of pneumonia. The last two figures are affected by the enormously high infantile mortality (about 25 per cent. of all children die within the first year of life !). But even deducting the first year, the figures read like this : Pulmonary tuberculosis, 215 ; bronchitis, 118 ; pneumonia, 674. Adding deaths from bronchitis and pneumonia together and comparing them with those due to pulmonary tuberculosis, one arrives, for 1932, at the following table :

MORTALITY PER 100,000 INHABITANTS, 1932.

	Cairo.	*Alexandria.*	*Provincial Districts under Medical Survey.*
Pulmonary Tuberculosis	764	454	1,639
Bronchitis and Pneumonia	5,111	3,331	14,222
Relation P. Tub. : (Bronch.+Pn.)	1 : 6·68	1 : 7·33	1 : 8·68

One would think that these figures must be grossly wrong. Such relations exist nowhere in Europe, and experience in daily life soon gives the impression that a comparison between Egypt and Europe should reveal an overwhelming prevalence of tuberculosis over other diseases of the respiratory organs in this country.

Lobar pneumonia is a rare disease. In Cairo the records for 1929 indicate 1·579 deaths from broncho-pneumonia, 1,943 from unspecified pneumonia, and 65 only from lobar pneumonia (p. 88, *Ann. Ret.*, 1929).

What is loosely termed pneumonia would therefore practically always mean broncho-pneumonia. The latter might be expected to be relatively rare for two reasons : the average age of the Egyptian is about twenty years below that of the European, and it is the advanced average age in Europe which accounts for the high death-rate from broncho-pneumonia there. Secondly, the climate of Cairo is the last in the world that might be suspected of favouring broncho-pneumonia. However insignificant the climatic influence on pulmonary tuberculosis, it is nevertheless difficult to imagine a place in Europe where the conditions are less conducive to bronchitis or broncho-pneumonia than in Cairo.

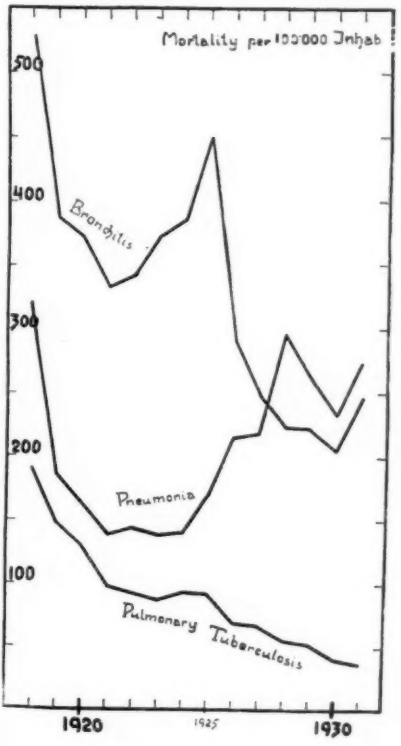
It thus seems manifest that the available statistics are definitely misleading. Some causes of the inaccuracy are easily perceived. Diagnostic errors are responsible to a lesser degree than the (sometimes pardonable) desire to conceal the true cause of death. The secrecy of a death certificate is not sufficiently safeguarded, and the practitioner intending to certify a death as being due to consumption has to face the determined opposition of the mourning family in whose hands he has to place the document.

Irrespective of many other possible causes of the inaccuracy in the statistical material, the conclusion seems inevitable that reliable figures are not available at present. An unknown, but considerable, fraction of deaths due to tuberculosis ranks under other names of respiratory diseases, leading thus to a false ratio of consumption to the group of pneumonia *cum* bronchitis.

Yet there is an opposite fact which seems to justify rather the accuracy of the statistical data—the decline of the tuberculosis death-rate. Since 1918, when a statistical survey was issued first, the curve of tuberculosis mortality falls rapidly and without interruption almost at an even rate. Or let us be more cautious in the interpretation of the facts : since 1918 the doctors of Egypt have continued to certify less and less deaths as being due to tuberculosis. As may be gathered from the diagram (p. 65), the curve of deaths from pulmonary tuberculosis per 100,000 inhabitants has, between 1918 and 1931, fallen from 191 to 37. What is the cause of this phenomenon ? Is the diagnostic capability declining ? Considering that possibility with an open mind, I feel convinced that the contrary is true. I have no doubt that a high percentage of deaths from tuberculosis is not diagnosed correctly, but I cannot believe that this inaccuracy has been increasing persistently. On the contrary, the growing use of diagnostic X-ray plants and the opening of laboratories where sputum is examined gratis are bound to have improved the diagnostic accuracy since 1918. Therefore there must be either a real decrease in the incidence of tuberculosis or else there are other reasons for which more and more deaths from con-

sumption escape registration. Actually it is my impression that both explanations are in some degree valid.

As to the decrease in notification, there may be a change in the class of patients who are admitted to the hospitals, for certainly the hospitals register their deaths more correctly than the general practitioners. It is also affirmed that the simple registration of deaths without diagnosis, covering the total of the 16 million population, has become more complete during recent years. According to an interesting paper by Dr. A. M. Kamal, lecturer in vital statistics of Cairo University, the "registration of both births and deaths was up till recently—if not up till now—inaccurate." It is possible that a relative decrease of consumption may result from this fact. It must further be realised that a high percentage of medical death certificates are written by a public health officer of the districts who sees the case for the first time after death and has to make a diagnosis from the history which the relatives of the deceased supply. Where there is no doctor, the undertaker of the village has to announce the death, a man who mostly cannot read or write. These factors, and probably others which I propose to analyse in detail in a subsequent paper, may tend to reduce the recorded deaths from tuberculosis below the true number.



There is nevertheless reason to believe that a real decline of the disease exists as well. If Clot Bey, a distinguished French physician, in the years from 1825 to 1840 observed only a "very small number of natives showing signs of phthisis," then it may pass for an established fact that in the first half of last century consumption was a rare disease. This conclusion is corroborated by the fact that the physicians contributing the medical articles to the standard work of Napoleon I. on his Egyptian expedition do not mention consumption (Levy).

On the other hand, tuberculosis is, in our times, undoubtedly very

common in Egypt ; no physician living here for a short while could overlook its frequency. Again, the official statistics which exist since 1918 tell us of a fall in the tuberculosis mortality which is unparalleled in its rapidity by any European State. Between 1920 and 1930 it declined from 129 to 41 ! The old statistical table of Levy[3] covering the period from 1901 to 1909 allows of a rough comparison which remains convincing even if a great allowance is made for incomplete recording. In 1905 the population of Cairo was equal to that of Alexandria in 1932 (about 650,000) ; in Cairo there were, in 1905, 1,379 deaths from tuberculosis recorded, whereas the figure for Alexandria for 1932 is only 585. Statistical differences of such magnitude are conclusive. They prove *a real and rapid abatement of consumption in Egypt during the last thirty years.*

It may seem paradoxical to doubt the accuracy of the statistical recording and yet to draw such an unexpected and important conclusion from it. The apparent contradiction is reconciled by a simple consideration of the absolute figures. In 1931 the recorded mortality from tuberculosis is 37, whereas it is 521 for pneumonia and bronchitis. Suppose that there is only one-third of all cases of tuberculosis correctly registered and that the official figure should therefore be multiplied by three while twice that amount ought to be deducted from the pneumonia group, even then 111 cases only of tuberculosis stand against 447 cases of pneumonia and bronchitis. The recorded figures for tuberculosis may, in reality, represent, say, only half, even only a fifth, of the truth, yet this (more or less constant) fraction will truly follow the decline of the disease during the course of the years. In other words, the falling tendency of the curve is correct and is a most interesting fact in itself ; but it is impossible to say on what absolute level that falling curve should be plotted above the one which can be drawn from the official records.

Here is the answer, then, concerning tuberculosis in Egypt : the disease has been endemic here since Pharaonic times ; not only in Egypt, of course. It must have co-existed among the other peoples of the Near East, and it is surmised that from here it spread, together with their civilisation, to Crete, Greece and beyond. It continued to be an endemic disease in Egypt through the ages, forming, perhaps, epidemic waves now and again of which no record exists. But the last wave of this type (or is it the only one that ever occurred ?) is being witnessed in our times. It must have had its crest not far from the beginning of this century, and what appears now as a falling curve on the paper is merely the expression for the ebbing of that wave. Again, it has certainly not run as low yet as statistical figures appear to indicate. Consumption is, at present, far more common in Egypt than it is in European countries, but it is left to the retrospective work of the

future to arrive indirectly at more accurate figures than those which are now available.

The decline of consumption in Egypt is of particular interest in view of the fervid discussions as to the causes of that decline in Europe. The Egyptian example seems to support the contention of those who lay stress on the spontaneous retrogression of the disease (through selection) and are willing to allow but little credit to all measures that have been taken in Europe to combat it (B. Lange, Andvord, Flatzeck-Hofbauer). The experience of European countries that the decline began everywhere before prophylactic steps were taken finds an outstanding confirmation in Egypt. As will be seen later, the first and only sanatorium of the country was opened as late as 1926.

Those who see the main cause of the decline of tuberculosis mortality in a rising standard of life in Europe (M. Greenwood, G. Wolff, L. Hersch) will find it difficult to apply their hypothesis to this country, where the rural population has not undergone any change in its standard of life for the last thirty years, and where the poor quarters of the towns are just as unhealthy (and as picturesque !) as they must have been a hundred years ago.

It is further remarkable to see how the curve begins in 1918 with a sudden drop, suggesting that a peak would appear if the curve could be continued backward during the years of the war. This peak between 1915 and 1918 is familiar to us from European curves, where it represents the years of deprivation. But in Egypt those years were blessed with prosperity like none before or afterwards.

Such observations (which I intend to analyse in a later paper) are apt to make us hesitate in attributing the decline of tuberculosis to one or another factor exclusively ; selection, welfare, prophylaxis and other causes have all accentuated the decline, but it is their relative importance which is under discussion (Gottstein, Burnet, MacNalty ; the brilliant book of Flatzeck-Hofbauer is specially recommended for studying the subject). The case of Egypt is apt to contribute many interesting points to that controversy.

B. Prophylactic Measures.

Private initiative took the first steps against tuberculosis in Egypt. In 1903 a committee of some European doctors and philanthropists formed a league in Alexandria which has been doing useful work from the time of its foundation up to this date. At present there are three dispensaries attached to this league, all situated in densely populated quarters of Alexandria. Unfortunately none of them is equipped with either X-ray

plant or pneumothorax apparatus. A fourth dispensary fitted with both X-ray and pneumothorax apparatus was opened by the Anglo-Swiss Hospital two years ago and is being visited by the present writer. The dispensaries of the league have considerable charity funds at their disposal (about £2,000 p.a.) which enable them sometimes to repatriate tuberculous foreigners; a great benefit for the latter, considering that they have no chance to find a free sanatorium bed in Egypt. In the Anglo-Swiss Dispensary I have to be contented with a small percentage of fresh cases in which a pneumothorax can save the situation or where screening and a further diagnostic analysis leads to some practical result. As a whole, the value of a dispensary in Egypt cannot be compared with that of a similar establishment in Europe. Nine-tenths of the population here live in desperately overcrowded conditions and in a state of poverty which cannot be alleviated by a few ounces of cod-liver oil and some well-meant hygienic advice. The dispensaries have no sanatorium beds at their disposal for the distribution of cases showing fresh activity. The European type of patient who has had his rest in a sanatorium, who has learnt there how to live and who only needs the after-care of a dispensary, is practically non-existent in this country. I have pointed out in the *Egyptian Medical Journal*[16] that the further opening of dispensaries is a waste of funds, while the much more urgent need of sanatorium accommodation has yet to be met.

The merit of having opened a sanatorium rests with the Ministry of Wakfs. This administration purchased a big old hotel (Al Hayat) at Helwan and turned it into a hospital for chest diseases under the name of Fouad Sanatorium. It was opened in 1926, and from then until 1932 was attached to the Medical Section of the Wakfs Ministry under the wise and energetic administration of Dr. Salem Bey Hindawy. He first called Dr. R. Burnand from Leysin to take charge of the institution. The pioneer work of this distinguished specialist was a guarantee of success. Demands for admission began to pour in, and it soon became obvious that the 150 beds which were available at first could not satisfy a minute fraction of what was needed.

In 1929 Dr. R. Burnand returned to Switzerland and the writer had the honour of succeeding him. Meanwhile the building had been partially reconstructed and could now provide accommodation for nearly 400 patients, but financial difficulties of the Ministry prevented the full use of the available beds at first.

The Fouad Sanatorium cannot provide any comfort in the European sense, but it is fully equipped with all that is needed for efficient treatment of chest cases. I was fortunate in having the collaboration of a staff of eight Egyptian assistants, most of them excellent men. In 1932 the Sana-

torium changed status, and became attached to the Public Health Department. I declined the renewal of a contract and no European director has been appointed since that time.

The Public Health Department of Egypt began the anti-tuberculosis work in 1929 by opening two dispensaries, one at Cairo and one at Mansourah. Three more have been opened since then. Dr. A. Latif Hassan, who is in charge of the dispensary at Mansourah, describes the difficult work of such an institution with touching sincerity : " Cases that are hopelessly diseased or very toxæmic are to remain at home, and a relative is sent to take the antiseptic or any symptomatic medicament, when necessary. Cases suffering from less hopeless lesions are told to attend the dispensary for treatment. The patients of this group are now and then visited by the medical officer of health of their locality, but we do not hear much of them except when they die ; the dispensary is notified of their end. It is unfortunate that in the provincial dispensary these patients form the bigger group ; in Mansourah, for example, they are 75 per cent. of the total." A sanatorium of the Public Health Department is now under construction not far from Cairo.

All that has been done against consumption in Egypt certainly falls very short of the most urgent needs ; but I do not wish to imply that it would always have been easy to do more. This country differs from Europe in too many respects to allow of a simple comparison, and if measures are copied thoughtlessly from Europe, where they may have proved useful, they may turn out to be worse than useless here. It is nevertheless certain that the construction of some more sanatoria is a most urgent need in the present state of prophylaxis.

The spontaneous decline of tuberculosis does not, of course, relieve the authorities of the obligation to build sanatoria. From a humane point of view the suffering of those who have been engulfed by the epidemic wave is too great to be left without the help of society. And as for the prophylaxis, the experience in Europe leaves no doubt that the falling curve will not reach zero, but will arrive and keep stationary at a certain level corresponding to the incidence of tuberculosis as an endemic disease. This figure is not predestined once and for ever ; it is largely influenced by the extent to which it is possible to segregate infectious cases in suitable institutions. To-day's sanatorium accommodation would be totally inadequate even if the number of consumptives declined in the near future to an irreducible minimum.

C. European Patients in Egypt.

If the old dispute on the value of climatic cures were settled it might be easy to give definite advice concerning Egypt as a health resort for

European tuberculous patients. Those who affirm that a patient should cure where he has to live after his restitution will *a priori* dissuade anyone from undertaking a long and costly voyage to the valley of the Nile. Those, on the other hand, who attribute to different elements of a given climate a potent influence on the course of pulmonary tuberculosis will probably ask how the climatic factors of Egypt are characterised, and will then form their advice accordingly for each given case. The writer has never been convinced by either doctrine. The only two specialists who have written on the subject from personal experience in Egypt arrived at contradictory conclusions. Dr. Levy, who was in charge of the dispensaries of Alexandria until his death (1935), thought that the climate of both Upper and Lower Egypt is not favourable for chest cases, and preferred to send patients to Europe for two or three years, if the means permitted the journey. It may be doubted whether anyone who spent all his life in Alexandria can have sufficient personal experience on the course of tuberculosis in other countries to draw such comparisons. The case is different with Dr. Burnand, who had gathered twenty years' experience at Leysin before he came to Egypt. He formed the opinion that the results obtained at Helwan are "often remarkable and rapid, comparable in every point to those observed in the best climatic stations of Europe." If it is not possible to arrive at a final conclusion about the influence of climatic conditions on pulmonary tuberculosis in spite of the numerous statistics on cure results that have been published, the reason lies in the personal factor of those who collect the material for the statistical figures. Granted even a determined effort to be as fair as possible in the interpretation of their own cure results, it remains impossible for different workers to classify and judge their material with such impartiality that a conclusive comparison between the results is ever possible. But in the case of Dr. Burnand there is the same judge who compares his experience of Leysin with that at Helwan, and it is remarkable to see that the extreme climatic differences between the two stations did not impress him as producing different results. It is a lesson which should be remembered by those who tend to exaggerate the influence of even minute differences in the climate of two localities.

It is my belief that the influence of the climate on pulmonary tuberculosis is negligible in nine cases out of ten ; consequently I cannot advise any patient with limited means to undertake the journey to Egypt. On the other hand, where financial questions are unimportant, the suitable case may improve in health through the pleasures of a voyage to sunny climes, whether it be Egypt, Switzerland, Madeira, or wherever the personal choice may fall. Anyone who has been able to live for a whole winter in the high altitudes or in the south and then makes a comparison with a winter in

England or in the lowlands of the Continent can hardly form a different opinion.

The suitable cases for Egypt cannot, above all, be those who need institutional treatment, as there is no European sanatorium in Egypt. The best suited cases may be those of a chronic, fibrotic type, with a normal temperature and a tendency to bronchitis or asthma. As the patient will have to stay in an hotel the physician should also consider his liability to spread infection.

Asthmatic attacks disappear instantly in the majority of cases as soon as the patient arrives in the dry, bracing winter air of Cairo or Upper Egypt. In fact, pure bronchial asthma seems to be a much more convincing indication for a journey to Egypt than pulmonary tuberculosis.

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REMARKS ON THE CLINICAL ESTIMATION OF RESISTANCE IN PULMONARY TUBERCULOSIS

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In attempting to bring this wide subject within the limits of a short paper, it will be necessary to confine my remarks to the practical and purely clinical aspects of the subject, using the term "resistance" in a loose but clinically acceptable sense. Avoiding, then, the wide implications of the word "resistance," which embraces the interrelations of allergy, immunity, character, and age of tissue—a host of debatable points—I propose to apply the expression in this popular sense, and having regard to its direct effect on the patient's outlook or prognosis.

By this I mean something more than a discussion of the chances of the tissues putting up a winning fight against the tubercle bacillus. The occurrence of living bacilli within calcified nodes induces the pessimistic conclusion that the tissues seldom win outright. The protracted march of tuberculosis, *par excellence*, the slow motion picture of a disease process, with its frequent compromises between invading organism and resisting tissue, broken often by some intercurrent circumstance permitting escape of bacilli from investment and further infection—all these considerations contribute to this view. On the other hand, when we regard the universally high rate of infection, and turn to the records of adult autopsy findings, as well as the results of experimental infections, we can state definitely that in large measure the tissues do frequently win their fight.

Our problem now is to decide at the bedside what is going to happen to the patient in the near future, and to do this we must rapidly review a series of facts and findings in relation to his case.

The clinical flair of the older generation of physicians was a valuable possession acquired by long experience—a subtle something which the possessor sometimes could not explain fully. Close observation and a long acquaintance with the processes of disease, especially if the observer possesses a wider clinical ground than that of the narrow specialist, can usually justify an accurate forecast, and if we add to these attributes a sound knowledge

of the pathology of tubercle, in its various types and manifestations, and employ with discrimination certain clinical tests, we may even attain the level of an exact scientific assessment.

The dictum that the success of treatment depends upon the presence of a cavity is just as misleading a generalisation as the idea that a positive sputum considerably jeopardises the chances of an ultimate recovery. Sanatorium statistics notwithstanding, every clinician of experience has seen cavities completely disappear with or without collapse therapy, or dry up and the sputum lose its bacilli, and this is confirmed constantly by serial X-ray examination.

Let us now summarise very briefly under familiar headings the information from which we may draw our inferences. A full and critical enquiry would unduly prolong this paper, so one's remarks will be confined to a short sketch of points of practical value in the assessment of a case when first seen.

1. *The History: Mode of Onset.*—It is a commonplace that cases commencing with hæmoptysis have a better outlook than those commencing insidiously or with catarrhal symptoms, and that those following some debilitating illness—*e.g.*, influenzal pneumonia, enteric fever, etc.—have an outlook less favourable still. The first statement rests on a somewhat insecure basis, since only 7 to 10 per cent. of cases commence with an hæmoptysis, while 80 per cent. are insidious or catarrhal at the onset. It is more likely, however, that hæmoptysis determines an examination much earlier than the insidious or catarrhal modes, and this affords an earlier control. Occupation plays a part in so far as it often decides the amount of time the subject can devote to "curing," but with proper sanatorium facilities the degree of revolution brought about in the patient's mode of life often decides in favour of the poor and against the rich. Risky trades involving pneumoconiosis, and especially silicosis, are bad factors in the ultimate prognosis.

Amongst preceding illnesses a history of pleurisy is a fact of major importance, because we now regard pleurisy as merely a stage in the evolution of pulmonary tuberculosis. Moreover, the possibility of pleural adhesions which may negative efficient control by collapse therapy, should it become necessary, makes pleurisy an unpleasant factor in the future of the case.

Long-standing so-called bronchitis of elderly people should always call for careful investigation when tuberculosis breaks out in younger members of the family. The unsuspected chronic tuberculotic masquerading as a bronchitic has often brought dangerous infection to younger members of his or her household.

Severe influenza, especially with pneumonic signs, enteric fever and malaria, are bad predecessors to pulmonary tubercle.

2. *The Family History.*—Points of prognostic importance in the family history relate to the vexed question of heredity. The old idea of the inheritance of susceptibility to tuberculosis, once so firmly established before the advent of Koch, still lingers in the layman's conception of the disease, and largely colours even stereotyped medical teaching. The opposing doctrine, defended by Sanarelli, who postulated the inheritance of resistance under modern conditions of urban life, seems to be gaining ground amongst clinicians. Moreover, it brings tuberculosis into line with the majority of bacterial infections, and it explains in large part the differences in susceptibility and mortality in racial groups living under the same urban conditions (*e.g.*, New York). Resistance against tuberculosis depends on *contact* with the tubercle bacillus, and can only be acquired by the tissues' successful struggle in limiting the advance of the germ. Elsewhere[1] I have summed up in review the facts and opinions which seem to me to suggest that the inheritance factor, though often complicated by home infection and contact with tuberculosis, means little more than the passing on of a slight degree of resistance acquired by the successful conquests of previous generations. It is probable, then, that children of tuberculous parents do not inherit a predisposition, but rather an increasing measure of resistance to the disease. The importance of the family history lies mostly in discovering the degree and length of possible exposure and in revealing the kind of *environment* in which the family life has been carried on.

3. Close attention given to the appearance of the patient, to his build and general make-up, frequently yields evidence of the kind we want in greater measure than anything else. Intelligent inspection of the stripped patient will give the examiner a better estimate of his condition than any other part of the physical examination. Here and there one can pick out the unmistakable traces of the characteristic toxæmia* of tubercle. Loss of tonus (skin, vaso-motor and muscle) is an early sign, and clear evidence of the grade of poisoning. Easy sweating, myotatic irritability, muscle spasm and subsequent wasting with characteristic effect upon the musculature of the shoulder girdle, of the neck, of the spinal column, and even of the face, are indications of the extent and severity of visceral disease. The loss of lustre of the hair, the size of the pupils, and the pallor of the mucous membranes, of the sclerotics and of the nail-beds supply similar evidence.

* The term "toxæmia" is here employed to indicate a condition complex rather than an actual state of specific "poisoning," having full regard to the tenets of Krause and others who hold that the tubercle bacillus gives rise to no particular "toxin," but that symptoms are due to the absorption of degradation products of broken-down tissues.

Clubbing of the nails is of two types, aduncate and drum-stick, and these refer generally to the chronicity and grade of the pulmonary fibrotic or septic process. Variation in body weight above or below the patient's average, and in relation to the standard weight for his size, is valuable as an index of nutrition.

4. An estimate of the patient's temperament or nervous make-up is of great importance. The different kind of fight put up by the sanguine and by the dull or apathetic types is well known. The obstinate, self-willed patient, who is usually inaccessible to advice, is generally the "fool who never gets rid of his tuberculosis." Since success in the fight against pulmonary tuberculosis is in large measure the result of intelligent co-operation between physician and patient, much depends on the mental attitude of the latter, and on the bond of confidence established between them. Much has been written about the mental outlook of the tuberculosis patient. Its importance as a factor in the "cure" must never be underestimated. Complete frankness must characterise the relations of physician and patient; the latter must know exactly where he stands at the beginning of his breakdown, so that he may enter readily into a co-operative pact with his doctor. Except in hopeless and inevitably fatal cases I have never known any harm to come from a clear statement of the case to the patient. This will be tempered by common sense and the ability of the subject to understand the medical point of view. If the patient is tractable, is ready to learn the lines of the new life of the "cure," and will give his full support to his physician's teachings, he is bound to do well; if not, then "all the voyage of his life is bound in shallows and in misery."

5. In assessing the findings of the *physical examination* we should be careful not to allow the physical signs to weigh unduly in our final verdict. It is well to bear in mind that this examination merely covers the "locus minoris resistentiæ," and that we are dealing with a man suffering a wide range of symptoms and crippled in one or more organs. Our concern in prognosis is principally with the outcome of the struggle as it affects the *whole* man. Survival of the man with a more or less severely crippled organ is a common experience. Even total surgical elimination of one of the paired organs is consistent with cure. Our examination is intended to discover the type of lesion, the extent, its age, and the complicating or accessory organ involvements. Radiology is the natural complement of the usual physical examination, but no matter how excellent the quality may be, this must never be allowed to detract from the care and completeness with which that examination should be made. Radiologist and clinician must collaborate and dovetail their work; it is futile for one to claim the superiority over the other.

Here, then, with the clinical picture and the shadow tracing before us, we have a host of facts on which to make our final assessment. Here we can readily distinguish between the relative chances of recovery in the broncho-pneumonic type, the fibro-caseous, the fibroid and the miliary. The X-ray film will help to show up the early, clinically silent, thin-walled cavity, and will distinguish it from the more serious thick-walled old vomica. It will reveal the presence of unsuspected enlarged hilus glands, and will show the full extent of the spread of an apical hæmoptysis (" snow-storm effect ") and the presence of deeply placed interlobar effusions. It will help to settle the important question of the application of collapse therapy by indicating the extent, if any, and grade of contra-lateral involvement.

The collection and sifting of all these facts is a necessary preliminary to the selection of the line of special treatment which again will have some bearing on the final result. Thus the outlook for a case found suitable for collapse therapy is generally better than when such a short cut to the complete control of the lung lesion is impossible. Besides, collapse therapy being merely an extension of the fundamental principle of rest, its application does not preclude the use of other, more or less specific, remedies—*e.g.*, gold therapy and tuberculin. Every clinician of experience can probably recall the story of most unpromising cases, saved and permanently restored by the timely application of a therapeutic pneumothorax.

In spite of the mass of facts accumulated in these examinations we may yet lack the precise information we require regarding the humoral (serum) and tissue reactions towards the invading bacillus. To this end we may invoke the help of certain examinations of the blood and the urine as well as certain tuberculin tests.

6. The tuberculin test, using naturally a dependable type of antigen, reveals little more than the grade of allergic response on the part of sensitised tissues. This reaction can further be exploited to ascertain the minimum reacting dose (the so-called quanti-test) in order to find, when desired, the commencing dose for tuberculin therapy. Failure to react (anergy) to tuberculin is important evidence of danger in the presence of tuberculosis, but the converse, a high-grade skin reaction, does not *by itself* justify the conclusion that the fighting power of the tissues is good or bad, nor that a positive reaction to a certain dose of tuberculin indicates an active tuberculous process.

Regarded in the light of the clinical findings, a graded tuberculin test may be helpful in indicating the level of sensitiveness, and as an indication of the kind of reaction the tissues are putting up against the invader.

7. Further search for evidence bearing on the outcome of the struggle of tissue against invader has given us a number of tests of the blood which

are of decided value. The most useful are either the nature of a flocculation (precipitate) reaction of various reagents against the blood serum, or of the rate of sedimentation of the red cells in a column of whole citrated blood. Confirmatory evidence is also forthcoming from the study of a series of blood counts and dried films. The serum flocculation reaction permits of exact grading and measurement in the technique and by the photometer of Vernes—a test which certain large corporations, like the Union Parisienne d'Électricité in France, impose on all their employees. Enthusiasts claim that this test can with certainty distinguish the inactive from the active lesion. The test is certainly valuable ; its technique is simple, the range of personal error in making the estimate is small, and the only disadvantage is the expense of the delicate photometer apparatus. The sedimentation rate and blood-film examinations are essentially bedside tests, involve very simple and portable apparatus, and yield quick results and quite as valuable as the flocculation reactions. The speed of the red cell sedimentation is an unerring index of the amount of tissue disturbance, of protein break-up, and presumably lessened resistance. The indications of a single test are neither absolute nor specific for tuberculosis, but, in the absence of other obvious conditions, the blood sedimentation rate, examined from time to time, yields useful indications as to prognosis—that is, as to the state of resistance and the mechanism of defence. Plotted out, a series of such tests will show a curve characteristic of a type of falling, of sustained, or of increasing resistance, and indicate thus a corresponding prognosis (see Figs. 1 and 2).

Some help can also be got from an examination of the stained film. By observing and classifying the nuclei of a hundred neutrophile leucocytes according to the number of lobes, in the manner of Arneth or of Schilling, we can estimate the proportion of young to old forms, and deduce therefrom the character of the demand made upon the blood-forming tissues. Again, information in the same direction may be obtained by examining the percentage ratio of the lymphocyte, monocyte, and neutrophile in the stained film. Serial examinations supply useful indications according to the disposal of certain cells which have a bearing on the age and the development of the tuberculous focus. Certain general conclusions seem to have been established by a great amount of work on these lines. Thus Flinn,[2] amongst others, sums up the information as follows : Excluding other sources of sepsis, a neutrophile increase indicates an exudative lesion, and a steady rise of neutrophiles in serial examinations an extension of ulceration. A high percentage of lymphocytes is to be expected in a healing lesion, and a rising percentage is a satisfactory indication of continued progress towards healing. A relative increase in monocytes accompanies a proliferative

lesion, but a rising rate, if associated with a normal or low lymphocyte percentage, shows an extension or exacerbation of the proliferative process, or a complicating extra-pulmonary tuberculosis.

Taken together, then, the information obtainable from the blood film is merely suggestive ; it supplements and supports the results of the red cell

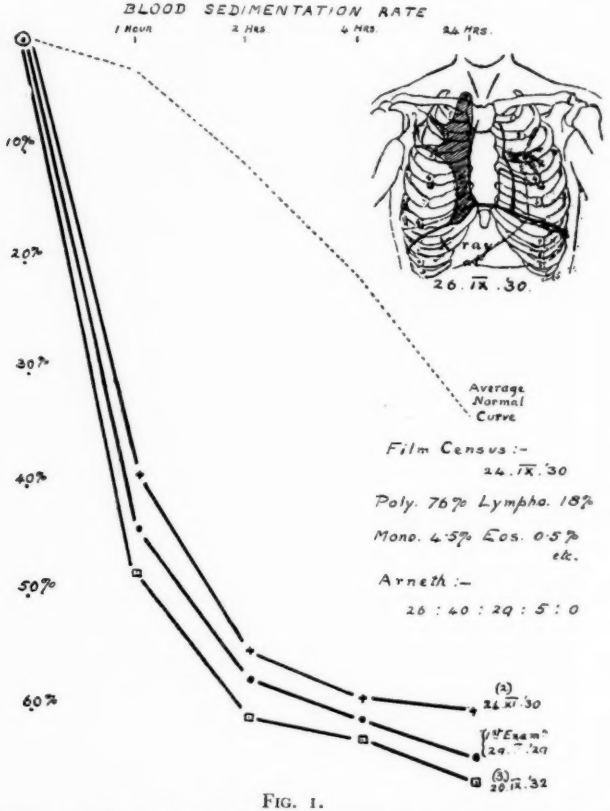

FIG. 1.

Miss E. Y. Blood sedimentation rate clearly indicates poor resistance at outset and failure to respond to treatment. First seen January 6, 1928. Artificial pneumothorax right ; incomplete collapse owing to adhesions. Spread to left lung. Collapse left lung February 17, 1932. Steady loss of weight (114¼ to 106½ pounds). Death, 1934.

sedimentation rate, but when observed serially, yields most definite indications as to the outlook of the case (see Fig. 3).

8. From a series of estimations of the vital capacity and on comparison with normal scales (*e.g.*, those of Dreyer, where stem-height, weight, and vital capacity are correlated) some further help in our assessment is possible.

9. The delicacy and exactness of the blood examinations already mentioned have largely detracted from the value of the diazo and urochromogen urine tests as indices of progressing disease.

10. In his examination of the stained film of sputum the bacteriologist

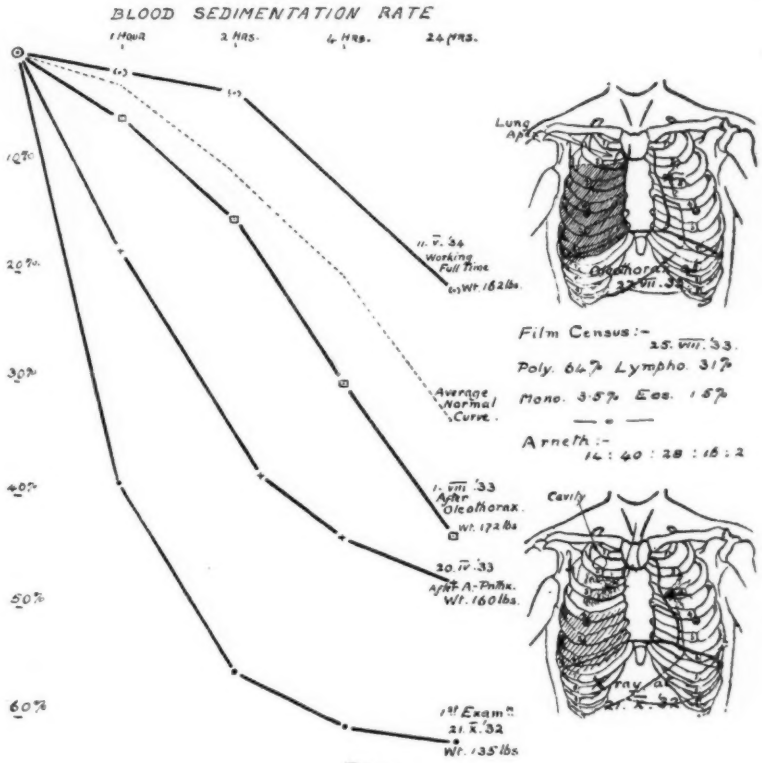

FIG. 2.

J. R., marine officer. Repeated pleurisy May, 1932. Hospitalised in Melbourne three months. Returned to South Africa October, 1932, with active and progressive tuberculosis (*vide* X-ray). Complete collapse of right lung fortunately secured, despite old pleurisy. Recurrence of effusion January, 1933. Oleothorax instituted gradually May-July, 1933, to prevent adhesions. Complete success. Steady gain in weight (135 to 172 pounds), in first year. Full work resumed May, 1934, with occasional heavy storm duty. Continues to date without symptoms, in good general health, and at full duty.

may be able to inform us as to the type of tubercle bacillus present, whether of bovine or human variety, whether young or old forms, whether of the R or of the S types on culture. But these details, though interesting, are largely academic, and assist but little in solving the important question of

the patient's prospects. Even the presence or absence of tubercle bacilli in the sputum, though of importance in the management of the case, is not a dependable factor in its outcome. This has long been a favourite basis for

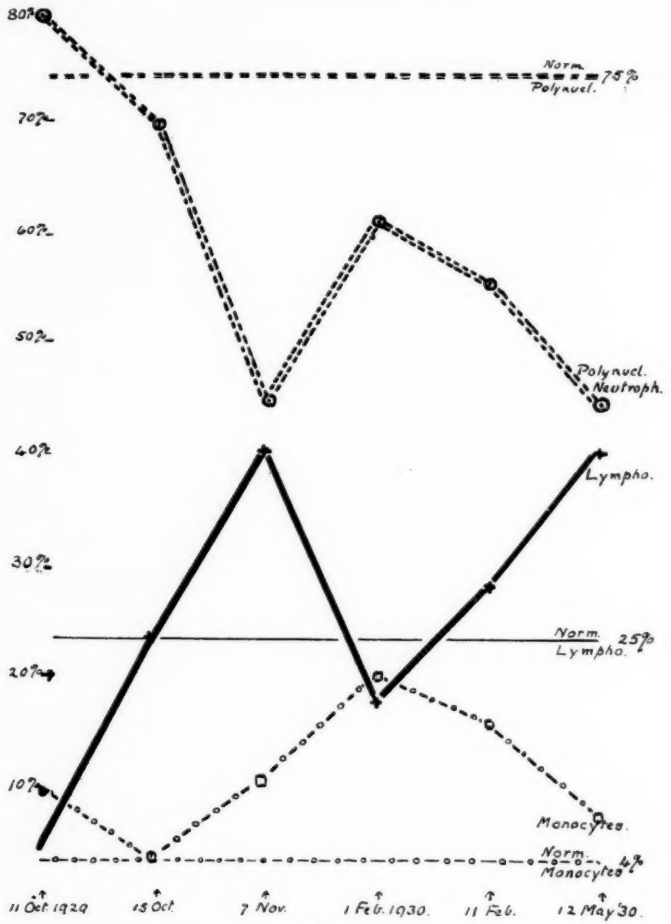

FIG. 3.

Miss F. Variations in resistance clearly shown by fluctuating values in blood polynuclear-lympho-monocyte ratios. Typical of tuberculosis of acute and progressive type (miliary). Death, 1931.

classifying the results of sanatorium cases, and the figures seem invariably to favour the bacillus-free, yet one feels that this finding must take its place in the line-up for review of all evidence required in the assessment of a given case

Enfin, it is clear that a true estimate of the prospects of the case cannot be made at one session. From no single line of examination can we get a clear-cut answer regarding the outlook towards ultimate cure. The wise and experienced physician, having at hand all the assembled facts, clinical, radiological, bacteriological, and biochemical, will study the more closely the mental, spiritual, and physical make-up of his case, and will watch his reactions, psychological and physical, before giving a considered opinion.

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SMALL LOCALISED ARTIFICIAL PNEUMOTHORAX

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IT is a general maxim that, when attempting the induction of an artificial pneumothorax, if, after repeated attempts in different situations, it is found that at the best only a small pocket can be obtained, holding say 200 to 300 c.c. of gas, and that only with a rapidly rising pressure, then it is useless to continue. It is generally accepted that in such a case the small pneumothorax so established is of no value, and that it is a waste of time to maintain it. Whilst this is undoubtedly true in the majority of cases, there occur occasionally a few instances where a localised artificial pneumothorax may prove of great value, the successful induction and maintenance of which being capable of saving the patient from the more serious forms of surgical interference.

One of the principal objects of artificial pneumothorax treatment is to promote closure of a cavity. In the majority of cases, when a proper collapse cannot be obtained, the small air pocket is usually obtained at the base of the lung, whilst the lesion is generally in the upper lobe, and is surrounded

by adhesions, which may prevent the basal pneumothorax from having any effect on the cavity. Occasionally, however, even one of this type may be of some value, acting in much the same way as a phrenic evulsion, by pushing the whole lung up to the apex, and so compressing and putting at rest the diseased area. We had one such case last winter in a young man of twenty-one. He had a cavity in the right upper lobe, the condition was very localised, and his general health pretty good. A basal pneumothorax was obtained after much trouble, and maintained, the air pocket holding about 300 c.c. at fortnightly intervals, always ending with high pressure. The cavity closed, all cough and sputum ceased, he gained much in weight, and left here in the early summer, apparently quite well.

If, however, with such a cavity, or system of cavities, localised at the apex, a small pocket can be obtained in this apical situation, the lower part of the lung being tied up with adhesions, then it is well worth while continuing, for a small pocket with high pressure in the immediate neighbourhood of the cavity will have a splinting action, and this, combined with the pressure effect, may cause it to close and heal. In such a case the local pneumothorax, though an intrapleural intervention, acts in much the same way as an extrapleural plombage, and much more satisfactorily, if successful, because no unabsorbable foreign body is used, and there are none of the dangers which may follow a major operation.

We have had two such cases under our observation last winter, and the following reports illustrate the value of this procedure.

Case 1.—The patient, a woman æt. thirty-five, arrived in Switzerland in September, 1933, with extensive disease in the left lung, with a system of cavities in the upper lobe. She had at this time also a pleurisy with effusion at the right base. An artificial pneumothorax was attempted on the left side, but without success. A phrenic evulsion, subsequently performed, brought a certain amount of benefit, though the cavities remained open, and the patient was never really well. Accordingly, as she was not considered fit enough to stand a big thoracoplasty, a pectoral plombage (De Winter's operation) was performed in November, 1934. This at first was of great benefit to the patient; the cough and sputum diminished, the temperature came down, and the weight increased. She was able to get up and go for short walks. In the new year, however, she began to fail again; the temperature went up, cough and sputum increased, and the weight dropped. It was then found that the disease was starting in the right upper lobe. She was kept in bed under observation for some weeks, but it became obvious from X-ray examination that a rather large cavity was developing in the right upper lobe. An artificial pneumothorax was then attempted in many situations on the right side, but the lower part of the lung was firmly adherent, owing to the previous pleurisy. Finally a small pocket was obtained just under the clavicle, at first holding only

50 c.c. of air, but in time we were able to develop it so that eventually she was able to take 200 c.c. every week, though the pressure rose rapidly. A skiagram showed that the pneumothorax was confined to the dome of the pleura, but it also showed that it had a direct effect on the cavity, which was definitely flattened.

At the present time the pocket is slowly obliterating, and it is now only possible to inject about 100 c.c. of air weekly under high pressure, but there is no doubt that this localised artificial pneumothorax has been of extreme value to the patient. The cough and sputum have diminished, and the blood sedimentation improved. Her general condition is better, and she is eating well, and gaining weight. A recent skiagram shows quite clearly that the cavity has been replaced by a fibrotic scar.

CASE 2.—A young man æt. twenty-one arrived in Switzerland in October, 1934, with a small localised lesion in the right upper lobe. He had a history of pleurisy with effusion on this side. The other lung was perfect, and his general condition good. A skiagram confirmed the presence of a fair-sized cavity in the lower part of the right upper lobe, the lesion being very localised. It was decided to attempt an artificial pneumothorax, and, as in the previous case, after many attempts, all that could be achieved was a small pocket from a puncture high up in the axillary line, which, when developed, held, at most, 300 c.c. of gas under high pressure. However, a skiagram showed that the cavity was compressed, and so fortnightly fillings were given. When he left here in early summer, a new skiagram showed quite definitely that the cavity was closed and healed.

Although the cases in which this treatment will be of value must naturally be limited, adhesions usually being present over the cavity, there are a few cases—especially those in which the cavity is a recent lesion, but has been preceded by attacks of basal pleurisy—when this is the interference of choice. The local artificial pneumothorax provides a method by which the cavity can be directly compressed, without resorting to serious surgical procedures, and without interfering even temporarily with the function of the healthy lower lobe. This is an important consideration when the other lung is not perfect. Moreover, it is possible in such cases to use much higher pressures than in a complete pneumothorax, because adhesions prevent the mediastinum from being pushed over and the heart displaced.

By this method it is sometimes possible to collapse thick-walled cavities which would not be influenced by a complete pneumothorax, and as this can be done with only a minimum of collapse of healthy lung, the patient is far less breathless than with a complete pneumothorax. Also, complications seem to be less frequent. We have had several other cases besides these reported without a single pleural effusion.

It always seems unfortunate that, when making a pneumothorax, it is necessary to collapse the whole lung in a case where only a small area of one lobe is diseased, as that lung never fully recovers function when re-

expanded, besides which the less strain thrown on the other lung the better. Such has been the success of localised pneumothorax in certain cases that experiments are now being carried out in Davos with the object of creating artificial adhesions of the lower lobe of the lung in a previously complete pneumothorax, so that the lower lobe will remain functional, and at the same time fix the mediastinum, and thus enable high pressure to be used in the localised pneumothorax now left over the apical lesion. If technique can be perfected it may prove a great advance in pneumothorax treatment, and will open the way for a far wider application of double pneumothorax.

However, a localised pneumothorax is a procedure which should not be undertaken by any except the experienced, as much damage can be done to structures in the chest wall near the clavicle, and high up in the axilla ; also it is possible to perforate the diseased area of the lung, and so cause a pyo-pneumothorax.

ERYTHEMA NODOSUM AND CERVICAL GLAND TUBERCULOSIS

THREE ILLUSTRATIVE CASES

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Erythema nodosum is very frequently, if not invariably, associated with a fresh infection by the tubercle bacillus.[3] Wallgren has found a strongly positive Mantoux reaction in 97 per cent. of a long series of cases, often accompanied by radiological evidence of a recent pulmonary lesion,[7] and concludes that in these cases erythema nodosum is the allergic manifestation of a particularly violent response to tuberculous infection. This infection may be primary or secondary ; if secondary, it may be due either to endogenous reinfection during a period of depressed resistance or to an exogenous reinfection or super-infection.

The three cases to be described were found in a series of over 200 cases of cervical gland tuberculosis. They illustrate both primary and secondary types of infection, and the peripheral course of the disease was clinically more or less apparent throughout.

Case 1.—Thomas M., aged 8. First seen in December, 1934, complaining of a rash on both legs, attributed to a kick on the ankle received

two weeks previously, together with progressive swelling of the neck. The skin lesions were typical of erythema nodosum. There was an adherent mass of glands in the right upper deep cervical (tonsillar) group, and the right tonsil showed in its upper pole a shallow oval ulcer 5×3 mm., with an irregular whitish floor. There was slight malaise and pyrexia, and the case was at first taken to be one of simple follicular tonsillitis. However, though the erythema faded rapidly, the ulcer reacted poorly to local treatment, and persisted for nearly three months, while the cervical glands assumed the chronicity characteristic of tuberculosis. It was decided to try tuberculin, which the writer has used with some success in cervical gland tuberculosis,[5] and after twelve months' treatment the glands have become quite small and mobile. The tonsil appears completely healed and shows no obvious disease.

Comment.

This is evidently a case of primary tuberculous infection by the tonsillar route, the allergic response to which was violent enough to produce erythema nodosum. It is suggested that the ulcer seen in the tonsil was a true tuberculous lesion formed at the site of bacillary invasion. The clinical evidence seems to warrant this view, though primary tuberculous ulceration of the tonsil is extremely rare. Webster[8] found the surface epithelium intact in eighty-six cases showing microscopic tuberculosis, but Dickey[2] has described a case clinically similar to the above in which tubercle bacilli were demonstrated beyond doubt.

Case 2.—Doris T., aged 23, domestic servant, in June, 1935, developed a swelling in the right side of the neck which grew to the size of a hen's egg. A rash came out on her legs, following which the lump in her neck became smaller. For several years she had been subject to sore eyes, but her health had been otherwise good, apart from an attack of pneumonia in childhood. She was found to be a well-nourished, healthy-looking girl, with mild phlyctenular conjunctivitis in the left eye. Fading lesions of erythema nodosum were present over both tibiæ. There was a group of three or four enlarged glands above the centre of the right clavicle, the largest of which was as big as a walnut, firm and fairly mobile, and some small glands were palpable in the right axilla. X-ray showed several large calcareous shadows in the right lung hilum and paratracheal region, but no evidence of any parenchymatous lesion. The patient was admitted to Seaham Hall Sanatorium, and at first became worse. The glandular disease spread upwards to involve the superficial cervical and lower deep cervical groups; there was severe bilateral phlyctenular conjunctivitis, with muco-purulent discharge and gross photophobia. Tuberculin treatment was begun, and after three weekly injections the eyes have almost cleared up and the glandular disease appears to be arrested.

Comment.

This case is one of old-standing tuberculous infection, as shown by the advanced degree of intrathoracic calcification, with a primary focus in the right lung. The probability is that, after remaining latent for a number of years, this focus became active during a period of depressed resistance. The usual paths of drainage to the hilum *via* superficial and deep lymphatics being blocked by disease of the broncho-pulmonary and tracheo-bronchial lymph glands, the infection passed outwards through adhesions between visceral and parietal pleuræ to the chest wall—a route suggested by Scott[4]—whence it tracked to the axillary glands, and from them direct to the supra-clavicular group. At this point allergy appeared, accompanied by erythema nodosum and exacerbation of the old phlyctenular conjunctivitis, and the spread of disease in the neck was temporarily arrested. Following this initial check a further extension occurred, which now appears to be under control by therapeutic tuberculin, eight months from the commencement of the attack.

Case 3.—George L., aged 12, first came under observation in 1932 with enlargement of glands above the left clavicle. An abscess was incised, after which the disease extended up the left carotid chain to involve most of the deep cervical group. Glands in the right posterior cervical triangle next became enlarged and also in the right axilla. None of these reached any considerable size, and the patient's general condition remained excellent. X-ray showed a healed primary complex in the left lung, with enlarged hilar glands and a calcified focus in the middle zone which may be taken to represent the initial infection, probably coinciding with the history of " pneumonia " at the age of eight months.

In May, 1935, there was an attack of erythema nodosum of a mild type, without any immediately preceding symptoms. There was no apparent constitutional disturbance, but the patient complained of pain in the cervical glands of the right side, which showed some transient swelling, but no further extension of the disease. X-ray showed no activation of the pulmonary or hilar foci. Tuberculin was given for four months, during which no further glands became enlarged and the patient continued to gain weight normally. He is now, in February, 1936, a well-grown, healthy-looking boy, with firm, mobile glands in both sides of the neck which appear to be undergoing fibrosis and calcification.

Comment.

Erythema nodosum occurred while this case was under observation. There was nothing to suggest an exogenous infection, tuberculous or otherwise, and therefore, in the absence of any other factor, its connection with the known tuberculosis may be presumed. It is probable that, as in Case 2,

a lapse in resistance occurred, allowing activity to develop in the right-sided group of cervical glands, and that renewed allergy resulted in the appearance of the erythema. The process in this case was in every feature milder than in the other two.

Summary.

Three cases are described in which erythema nodosum occurred during the course of tuberculosis of the cervical lymphatic glands.

In each case the tuberculosis appeared to be limited to the lymphatic system. There was no evidence of bacillæmia, and the erythema may be explained as an allergic phenomenon, connected in Case 1 with reaction to a primary infection, and in Cases 2 and 3 with an endogenous reinfection from latent tuberculosis of some standing.

Additional points are: in Case 1 the occurrence of what was probably primary tuberculous ulceration of the tonsil, and in Case 2 the association of erythema nodosum with phlyctenular conjunctivitis, itself regarded as an allergic tissue reaction to tuberculosis.[1, 6] The therapeutic success of tuberculin in all three cases may be noted.

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CONSULTATION

CASE

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This is the case of a man of twenty-eight, a merchant, who was admitted to a sanatorium with severe bilateral disease at the beginning of February 1935. Although he had been at work right up to admission, he proved to be febrile (p.m. rectal temperature for the first month was 100·2°). He had had a cough and expectoration for about six months and had lost some weight. Tubercle bacilli were very numerous and sputum two or three ounces a day. He had cavities at both apices with scattered infiltration lower down. Roughly three-quarters of the right and two-thirds of the left lung were affected. The lesions appeared, except for two big cavities at the top of the right lung, to be of the exudative type. The X-ray shadows were rather dense in patches and woolly.

He was put to bed and kept strictly at rest for four and a half months. For a long time the prognosis was considered to be rather hopeless despite the fact that his cough improved quickly and the temperature fell steadily though slowly. He gained exactly one stone in weight in his first nine weeks. When in July, 1935, he was beginning to be up for two or three hours daily, he gradually lost some weight, got increased sputum, less satisfactory temperature readings (up to 99·4° p.m. again), and a temporary slight inflammation, probably tuberculous, of the larynx. From early August till November he was given more rest and a course of sanocrysin.

By the end of December, 1935, when the consultation took place (eleven months from the date of his admission to the sanatorium), he was much better, quite afebrile, walking 1½ and occasionally 2 miles a day comfortably. He had lost his cough and almost his sputum. He had regained his weight and was maintaining it fairly well. The chief changes in his lung condition were the contraction of the right upper lobe with the cavities in it and the great improvement in the middle and lower lobes on that side. The lower limit of the excavated upper lobe had become

marked off by a thick, fibrous, sharply defined margin, which (serial radiographs showed) had risen about 2½ inches in ten months as the upper lobe contracted. Meanwhile, the left lung improved considerably, but mostly in its lower half. Cavities in the left upper lobe showed some improvement, but only slight fibrotic changes had occurred in it.

The problem was what to do next. Here was a patient who, under ordinary sanatorium régime, aided by sanocrysin, had progressed beyond expectations. He now had a comparatively healthy aspect and few symptoms. It was obvious, however, that his ultimate outlook was poor. He had relapsed once while in good environment and under medical attention. He was even-tempered and possessed of equanimity, apparently harassed by no serious mental perturbations and confident in his medical attendants. He showed a fairly full grasp of the situation at every juncture, and in particular seemed to realise the necessity for allowing ample time at every stage. Too frequently at a consultation adverse psychological factors, often all-important, have to be given grave consideration. But in this case, fortunately, they did not require much attention.

The first question to decide was: Would a continuance of an ordinary régime, even if he could put up with it, bring about further improvement, or was not active interference necessary? It was highly improbable that a proper recovery could be expected without resort to some form or other of collapse therapy. The next question, therefore, was: What form of collapse therapy should it be? And the next: When? Whatever was done, it would be unwise to carry out any form of collapse which would compress the lower lobes seriously or for long, particularly so if at any times both sides were being dealt with simultaneously.

The most obvious parts suffering from excavation were at the top of the right lung. Some would say a cavitated region which had shrunk upwards so much as this had done and which was so obviously surrounded by fibrosis had best be treated by apicolysis or by an upper thoracoplasty. Of the two, I should have favoured the latter, but felt that a postponement of any thoracoplasty was imperative to allow more time for further consolidation of the general health and of the healed infiltrated areas. I also came to the conclusion that a right-sided phrenic (which would have had to be a crushing in view of the probability of an upper thoracoplasty later) would not be likely to aid in the further contraction of the apical excavation. The right diaphragm had already become raised considerably by degrees and did not move at all extensively. Signs pointed to the fact that the lesions in the left upper lobe were those where activity had been most recent and the disease but poorly arrested. There were some arguments in favour of doing a phrenic on this side. If so, should it be a crushing only? Hemi-

paralysis of the diaphragm may influence the chances of inspired broncho-pneumonia at a base, possibly followed by fresh tuberculous infiltration, when a thoracoplasty is done on either side. My opinion, nowadays, is that the occasions when a phrenic operation should be undertaken as the first stage in collapse therapy should be considered exceptional. The next question, therefore, was whether pneumothorax on either side was worth attempting to see whether it proved selective. Some consider it advisable when extensive fibrosis is a prominent feature of an affected lung, or when the risks of harm from the complications of artificial pneumothorax (fluid, for instance) seem great, to proceed straight away to a thoracoplasty. I am always reluctant to do any kind of thoracoplasty before some trial has been made of A.P.T. Obviously, in this case, if artificial pneumothorax treatment compressed the lower parts of the lung and not the upper lobes, harm and not good would accrue, and it would have to be abandoned at once. It was decided, therefore, to attempt right artificial pneumothorax treatment at once and if this failed to try the other side.

P.S. (February 14, 1936).—A right artificial pneumothorax was induced on January 6, 1936. This allowed the upper lobe to contract even further, especially after a few adhesions had been cauterised under thoracoscopic control. The patient is now continuing to progress very satisfactorily.

CLINICAL CASES

PULMONARY INFARCTION

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Introduction.

The following case is thought worthy of record not only on account of its own exceptional interest, but also because it illustrates a difficulty in diagnosis which in our opinion is rarely sufficiently emphasised. It is well known that chronic pulmonary vascular congestion secondary to mitral stenosis can give rise to pulmonary signs and symptoms which may to some extent mask the causal lesion and be suggestive of primary disease within the lungs. Less well recognised is the even closer simulation of primary pulmonary disease which may arise when acute vascular congestion of the lungs occurs secondarily to left ventricular failure. Both in acute and chronic vascular congestion of the lungs diagnostic difficulties may be increased should infarction of the lungs complicate the clinical picture.

Case Report.

Mr. K. T., a Japanese film actor, aged forty-five, attended the Brompton Hospital on June 28, 1935, complaining of breathlessness and a persistent non-productive cough of some six weeks' duration. He stated that he "strained his heart" in 1932, and had as a result spent some two weeks as an in-patient in a provincial hospital (where he had been strictly dieted), and that following this illness, cough, breathlessness, and palpitation of the heart had continued for some weeks. He had had no other illness, and there was no relevant family history.

Clinically he presented the signs of chronic bronchitis with emphysema, together with tachycardia, and apart from extensive dental caries no other abnormality was noted. Sputum was negative for tubercle bacilli, and a radiograph of the chest (Plate 1) showed the following :

"*June* 28, 1935.—Diaphragm movements mainly thoracic. *Heart:* The cardiac margin is very poorly defined, and it appears to lie transversely. The heart is increased in its transverse diameter. *Lungs:* There is consolidation in both lung fields mainly around the hilum, extending out to the periphery. Appearances are unusual, and may be the result of congestion. The possibility of lymphatic spread of neoplasm cannot be excluded."

A week later clinically and radiologically there was no change in his condition. He was transferred and admitted to St. Mary's Hospital on July 20, having rested in bed during the intervening fortnight.

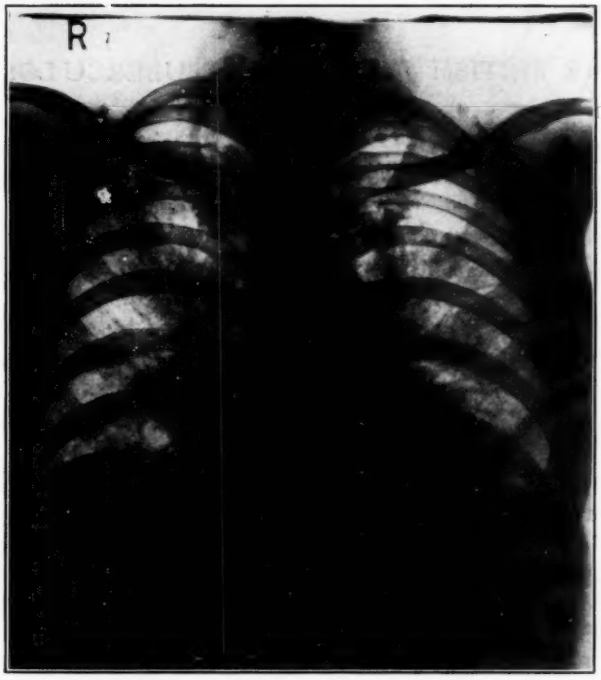
The dyspnœa meanwhile had increased, the cough become incessant, and productive of about 1 oz. of frothy sputum daily, and in addition he complained of nausea and vomiting after food, together with dysphagia. Examination showed that he had lost nearly a stone in weight since he first was seen, and that he was running a slight evening pyrexia.

The physical signs in the lungs were essentially unaltered; the heart, however, had enlarged considerably to the left, there was a regular tachycardia of 120 beats per minute, gallop rhythm, and a systolic murmur, not well conducted, could be heard at all areas. The blood pressure was 105/82 mm. Hg, although fairly marked peripheral arteriosclerosis was demonstrable in the brachial, radial and retinal vessels. Urine was concentrated and contained a trace of albumen. There was no peripheral œdema, and examination of other systems revealed no abnormality of importance. Sputum on four occasions subsequently contained no tubercle bacilli. Blood W.R. negative. Blood count: Hb=88 per cent. R.B.C.=4,400,000. C.I.=1·0. W.B.C.=7,800.

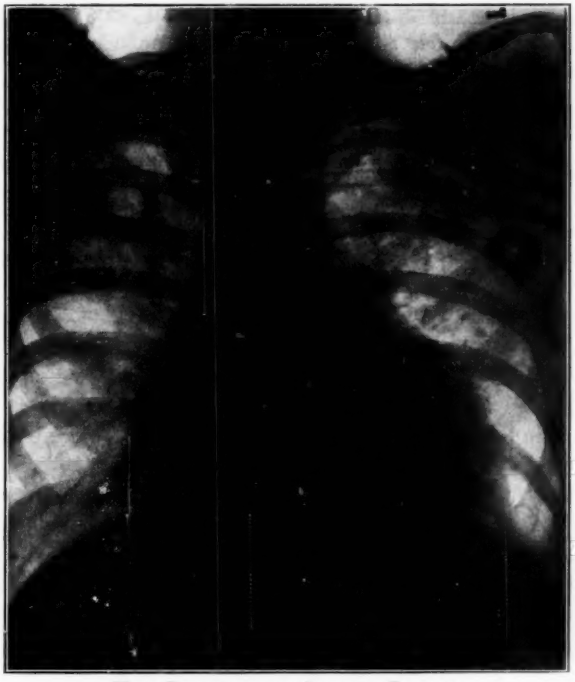
Electrocardiogram (August 1, 1935) showed the following abnormalities: Simple tachycardia. TI isoelectric. Right ventricular preponderance.

He was treated on general lines, including absolute rest, a light (mainly fluid) diet, alkaline expectorants, and sedatives, including morphia.

No marked change in his condition occurred until August 14, 1935, when he suddenly experienced continuous dull burning pain high in the epigastrium, vomited repeatedly, and was for a short time in a condition of shock. P.=148. T.=99·8°. R.=40. August 15, 1935: T.=101°. P.=136. R.=56. The urine output had fallen from about 30 to 15 ounces daily, and it contained considerably more albumen and a trace of sugar, while the sediment showed bacilli, red blood corpuscles and hyalo-granular casts. Cardiac dullness was now demonstrable to the right of the sternum, while the apex beat was in the mid-axillary line in the sixth intercostal space. Gallop rhythm was still present, as was the systolic murmur, but in addition a pericardial rub was audible on both sides of the sternum from



THE CONDITION ON JUNE 28, 1935.



THE CONDITION ON AUGUST 18, 1935.

the third intercostal space to the epigastrium. B.P.=110/80. Other systems unchanged. August 17, 1935: Precordial pain, dysphagia and vomiting were still present. The heart if anything was larger still, and the pericardial rub continued to be audible. P.=130. T.=99·6°. R.=46. B.P.=108/82. Orthopnœa and cyanosis were very marked, œdema of the feet was just evident, and a smooth tender liver edge could be felt at the height of inspiration. Other signs were unchanged. August 18, 1935: Patient free of pain. Dysphagia now accompanied by anorexia. Cough, sputum and dyspnœa unaltered. Pericardial rub inaudible, heart otherwise *in statu quo*. The lungs, however, had areas of impaired note and poor breath sounds at the left base posteriorly and in the right upper zone in front. Harsh râles and the signs of emphysema continued elsewhere as before. The liver was easily palpable and rather tender, and the peripheral œdema was more marked. Urine contained no glucose, but was otherwise similar to that of August 15, 1935.

Electrocardiogram: Closely resembling that obtained on August 1, 1935, save that TI was now just perceptibly inverted.

Radiological examination of the chest (Plate 2) showed:

"*Thoracic parieties:* No abnormality. *Pleural cavities:* Small left basal and right apical effusions. *Heart:* Gross general enlargement probably of all chambers. It is unlikely that a pericardial effusion is present. Hilar vascular shadows heavily exaggerated—(?) secondary to left auricular dilatation. *Right upper lung zone:* Almost certainly some tuberculous infiltration with a suggestion of early cavitation. Fibrotic changes also present in the left upper lobe."

The patient's course thereafter was that of rapid and progressive congestive heart failure, complicated by the appearance of a further area of impaired resonance with poor breath sounds in the left upper zone. There was little or no alteration in the cough, sputum, or dysphagia, and no further pain. Finally there appeared typical restless cardiac delirium with Cheyne-Stokes' respiration, the heart maintaining a regular tachycardia to the end. He died, following a terminal pyrexia of 103° F., on August 25, 1935.

Post-mortem findings may be summarised as follows:

Cardio-Vascular System: Marked general atheroma and arteriosclerosis. Calcareous plaques and ulcers in the abdominal aorta. Thrombosis with recanalisation of the right internal iliac artery. Thrombosis with recanalisation of the calcareous anterior interventricular branch of the left coronary artery. Recent complete thrombosis of the right coronary artery. Considerable fibrosis of the left ventricular muscle with a large aneurysm of the artero-lateral wall of the left ventricle in its upper part, filled with a firmly adherent and partly organised thrombus. Recent infarction and

necrosis of a large area of the lower part of the right ventricle with a considerable adjacent mural thrombus. Chronic adherent pericarditis of the pericardium overlying a large area of the left ventricle, recent adherent pericarditis over the infarct of the right ventricle.

Lungs congested. Multiple recent infarcts, the largest in the right upper lobe and in the left lower lobe. A minimal amount of old healed tuberculosis was present at the left apex. Small bilateral clear pleural effusions were present together with a few adhesions at the left apex.

Fibrotic old infarcts in the kidneys and spleen, while these organs and the liver showed chronic passive congestion. Recent acute peptic ulceration of the stomach. Peripheral dependent œdema well marked. Other systems not remarkable.

Discussion.

It would seem probable that in 1932 this patient had a coronary thrombosis involving the left ventricle with the eventual formation of an aneurysm of the heart. About this time embolism from the mural thrombus was responsible for infarction of areas in the spleen and kidneys, and possibly for the condition of the internal iliac artery.

He made sufficient recovery to live his normal life for three years, and then, presumably in May, 1935, his left ventricle began to fail, and by the end of June was giving rise to the symptoms which led to his attendance at hospital. Until August 14 clinically and radiologically severe left ventricular failure dominated the picture, while it seems likely that the symptoms of dysphagia and incessant cough were due to the pressure of the heart on the œsophagus and trachea respectively, especially since the pressure of the left ventricular aneurysm must have caused posterior displacement of the dilating heart.

On August 14 he had a thrombosis of his right coronary artery, and thereafter presented classical symptoms culminating in a rapid right heart failure and death.

The rarity of coronary thrombosis on the right side of the heart makes the case noteworthy. It is even more unusual for extensive thrombosis to occur on both sides of the heart, as was the case here. Remarkable also is the fact that at no time, either while the clinical picture was that of left ventricular failure, or later when pulmonary infarction was occurring as the result of embolism from the mural thrombus in the right ventricle, was hæmoptysis a symptom. Furthermore two electrocardiograms gave little or no indication of the state of the myocardium, though it perhaps may be noted here that leads 1, 2 and 3 only were taken, and it is possible that had three-dimensional cardiograms been obtained the true condition might have been demonstrated.

Radiological examination obscured rather than clarified the diagnosis, and the difficulty in interpreting radiographs of the chest in which congestion of the lungs is a feature, especially if complicated by pulmonary infarction, is illustrated.

Acknowledgment.

I am indebted to Dr. Thomas Nelson for his kindness in allowing me to transfer this patient to my own beds at St. Mary's Hospital and for permitting me to publish the case.

A CASE OF ACUTE PULMONARY ŒDEMA FOLLOWING ARTIFICIAL PNEUMOTHORAX

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As fatal complications of artificial pneumothorax are rare, it was thought that the following case might be worth recording.

The patient, a man of forty-six, was admitted to the hospital on September 9, 1935. The past history was unimportant save for winter bronchitis for several years.

On admission he had fibrotic disease, with some infiltration at both apices, more extensive on the left, with a small cavity in the sub-clavicular region, and an ounce of positive sputum. With general sanatorium treatment he made good progress, and after three months his right side was quiescent and he was on full walking exercise. The cavity at the left apex, however, was more apparent both clinically and radiologically, and his sputum was undiminished and still positive.

As it was essential for him to have a negative sputum before returning to work, it was decided to induce an artificial pneumothorax on the left side. This was successfully induced on January 3, and was carried on uneventfully until January 9. On January 10 his temperature rose to 99·6° F., he complained of some dyspnœa, and a small hydropneumothorax was found. The next day his temperature was 100·2° F., and the following day his dyspnœa increased, though the pressure in the pneumothorax was only −12–2. Beyond harsh breath sounds there was nothing fresh in the right lung, and he did not appear ill ; but on January 13 the dyspnœa was more urgent. On examination of the left chest there was no increase in the effusion, but fine râles could be detected throughout the right lung,

and he commenced to cough up copious frothy blood-stained sputum. A diagnosis of acute pulmonary œdema was made. He died the next day, January 14.

At the post-mortem the left pleural sac contained a pint of cloudy fluid, while the pleural membrane was injected and covered with flakes of lymph. The lung was collapsed, the apex alone being held out by two adhesions. The right pleura was normal, but the lung was heavy and œdematous. On section the lung exuded a large quantity of frothy serous fluid : there was no sign of pneumonia or broncho-pneumonia. There was some fibrosis at the right apex, but no evidence of any active disease. The left apex had a cavity about $\frac{1}{3}$ inch diameter filled with caseous material. The heart was normal, and there was no evidence of any disease in liver or kidneys. The clinical diagnosis of acute pulmonary œdema was confirmed.

The only explanation we can give is that the acute œdema was a reflex phenomenon caused by the pleurisy in the left side.

I am indebted to Dr. G. Oliver Hempson, the Medical Superintendent of this hospital, for permission to publish this case.

INFILTRATING TUBERCULOUS LESION OF CHEST WALL

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The patient, a youth of 19, had a right artificial pneumothorax induced in hospital in December, 1932, for pneumonic type disease involving the upper part of the right lung. He was acutely ill, but responded well, and his symptoms cleared up completely.

In February, 1933, his temperature rose suddenly to 104°, and fluid was found in the A.P. cavity. Aspiration was not performed, and the temperature gradually settled to normal. In May, 1933, he was transferred to a sanatorium, where his refills were continued. He made excellent progress and was discharged symptomless in December, 1933, to attend as an out-patient for his A.P. to be maintained. His right pleural cavity was still half full of fluid, which had not been interfered with.

Shortly after discharge he again became acutely ill, with temperature rising to 103·5°, and was re-admitted. X-ray examination revealed gross spread of disease in the previously clear left lung, again of the pneumonic

type. It was decided to allow his right lung to expand with a view to attempting a collapse of his left lung later.

The disease in the left lung continued to spread. Despite the fact that no further refills were given, there was very little evidence of any expansion of the right lung, presumably owing to the thickness of the visceral pleura. In the pleural fluid, which was purulent to naked-eye examination, no T.B. could be detected microscopically, and cultures for pyogenic organisms remained sterile.

In January, 1934, the disease in the left lung was advancing rapidly, and he developed a broncho-pleural fistula on the right side. T.B. were then found in the pleural fluid, but it still remained sterile to all pyogenic organisms.

Shortly after this he started to complain of discomfort, accentuated by coughing, in his right chest wall just below the nipple. For some time nothing could be found to account for this pain, but later a small area was detected close to the right nipple, where the chest wall was apparently thinner and softer than the rest. There was no involvement of the skin nor any induration of the tissues. This area slowly increased in size until a distinct bulge could be observed whenever the patient coughed. The process continued until the chest wall at this point appeared to consist of skin only.

Considerable pain was caused by coughing, but was relieved to a great extent by the application of an elastic belt with a pad controlling the bulging area. Eventually in May, 1935, the skin of the chest wall gave way and the fluid contents of the right pleural cavity were evacuated. A large area of skin then sloughed away, revealing a granulating discharging area involving the underlying muscles.

It was then found that the same phenomenon was occurring in the next lower intercostal space, and the whole cycle of events was repeated until there were two granulating areas which gradually spread and then coalesced, the combined area eventually measuring about 4 inches in diameter. The ribs did not appear to be affected. The pleural cavity continued to discharge large amounts of purulent fluid, necessitating frequent changes of the dressing.

Later, another intercostal space was involved, but the patient died in August, 1935, before perforation took place at this point.

Although no pathological examination of the diseased tissues was made, there can be no doubt that the above case demonstrates a very unusual complication of fluid in an artificial pneumothorax cavity—namely, a tuberculous involvement of the pleura spreading into and destroying the soft tissues of the chest wall.

It is noteworthy that, even after free drainage had been established for some time, the disease continued to involve and to perforate fresh areas of the chest wall.

A FATAL CASE OF SPONTANEOUS HÆMO-PNEUMOTHORAX

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A CLERK aged 35, who had never been ill before, had a loose bowel action and collapsed with a severe pain in the right side of the chest. After this he improved somewhat for two days and was then admitted to hospital looking pale and very ill. Pulse 130. Respirations 28. He had pain in the right side of the chest on breathing. He was restless and short of breath.

On examination the physical signs were quite definite. The apex beat was in the fifth intercostal space 5 inches from the midline. The right side of the chest was dull to percussion. The breath sounds were amphoric. The coin sound and succussion splash were present, indicating a hydro-pneumothorax. This was confirmed by X-ray examination.

Although he said he had always been pale, it was felt that the anæmia was so marked that it must be due to internal hæmorrhage. While under observation for signs of increasing pallor and pulse rate, he suddenly collapsed and died shortly after admission.

Necropsy.

The right pleural cavity contained several pints of fluid blood and some air.

There was a minute tuberculous cavity at the apex of the right lung, but it had not ruptured. There were no emphysematous blebs. On the anterior surface of the right upper lobe there was a roughened area of pleura and bleeding and escape of air had apparently originated here, but there was no gross macroscopic change. Apart from the minute cavity there was no other evidence of tuberculosis in the right lung. The other lung and the rest of the body were quite normal.

Commentary.

Traumatic causes of hæmopneumothorax are seen fairly often, and it has recently been shown by Cooke[1] that trauma sufficient to damage the lungs and pleura may yet leave the parieties unharmed. Recorded cases of spontaneous hæmopneumothorax, however, are very uncommon. More cases have probably occurred than have been reported as such since before

X-ray examinations were made cases described as hæmorrhage pleurisy were probably cases of hæmopneumothorax but the pneumothorax was missed.

Jones and Gilbert[2] surveyed the literature of the past fifty-five years and found records of nineteen cases of spontaneous hæmothorax in apparently healthy people, and Koral,[3] who added cases with frank tuberculosis, found thirty-four cases since 1900.

In cases where frank tuberculosis is absent the etiology is somewhat obscure, but it appears to resemble that of spontaneous pneumothorax. The fact that a bloodvessel is ruptured must be regarded as a chance occurrence. It seems probable that a torn vessel associated with a ruptured emphysematous bulla or a torn adhesion will close in most cases when air escapes and causes the lung to collapse just as a bleeding vessel in the lung itself will sometimes close when artificial pneumothorax is induced. In rare cases, however, bleeding evidently continues. Koral[3] considers that the symptom complex in these cases is characteristic and consists of three stages.

1. The sudden pain and dyspnœa of spontaneous pneumothorax.
2. Temporary improvement lasting hours or days.
3. Recurrence of pain and signs of internal hæmorrhage with physical signs of fluid and air in the pleural cavity.

The present case shows these characteristics.

In only five cases of so-called idiopathic hæmopneumothorax prior to the present one have post-mortem findings been described. These were as follows:

1. Pitt's[4] case. Torn adhesion to an emphysematous bulla. No tuberculosis.
2. Rolleston's[5] case. Cause of bleeding not ascertained.
3. Case of Horsden and Piggott.[6] Torn adhesions to subpleural blebs associated with a puckered spinal tuberculous scar.
4. Fischer's case.[7] Torn emphysematous bleb at apex—the edges of the tear covered with coagulated blood. No emphysema or tuberculosis elsewhere in the lungs.
5. Case of Jones and Gilchrist.[2] Ruptured spinal emphysematous bulla, but the actual site of the bleeding was not found. No tuberculosis.

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MEETINGS OF SOCIETIES

JOINT TUBERCULOSIS COUNCIL

At the February meeting of Council twenty-five members were present. Dr. G. Lissant Cox was elected Chairman of Council for 1936, Drs. L. S. T. Burrell and S. Vere Pearson Vice-Chairmen, Dr. Ernest Ward Hon. Secretary, Dr. G. Jessel Hon. Treasurer, and Dr. D. P. Sutherland Hon. Auditor. The appointment of an Assistant Secretary was discussed.

It was decided to circulate to Medical Officers of Health and tuberculosis workers the biennial report of the Hon. Secretary, and also the report of the Contact Committee, presented by Dr. Hebert, and adopted after a brisk discussion. A most informative memo. on the Microscopical Examination of Sputum, by Drs. W. H. Tytler, H. G. Trayer, and Peter Edwards, was likewise adopted and will be circulated.

Dr. Brand was unable to be present, but had written the Secretary announcing the courses arranged for 1936. Dr. Peter Edwards would give three courses (April 21 to 23, June 23 to 25, and September 22 to 24), and a course at City Road Chest Hospital would be held from May 4 to 9. Drs. Peter Kerley and R. R. Trail would also be asked to give at a convenient time a series of demonstrations on X-ray interpretation in chest diseases. The question of a course in Surgical Tuberculosis would be considered later. Dr. Brand was heartily thanked for the arrangements already made and for those contemplated.

After the meeting members adjourned to the Hotel Russell, where the Council were entertained to lunch by their Chairman, Dr. G. Lissant Cox. Forty-two members and guests were present. Dr. A. S. MacNalty, Chief Medical Officer to the Ministry of Health, was the chief guest, and there were also present our previous Chairman, Sir Henry Gauvain, Dr. W. G. Savage, the President of the Society of Medical Officers of Health, and several medical editors: Dr. Horner (*British Medical Journal*), Dr. Morland (*Lancet*), Dr. Conner (*Clinical Journal*), Dr. Charles Porter (*Public Health*), Dr. Roodhouse Gloyne (*Tubercle*), and Captain Elliston, M.P. (*Medical Officer*). Other guests were Mr. Girdlestone of Oxford, Dr. Maitland Radford, Dr. Allen Daly, Professor Bedson, Dr. P. Hall Smith, and Mr. Lewis Elliston.

After lunch Dr. Lissant Cox proposed the health of Dr. A. S. MacNalty

and our guests. There were present, he said, all the Medical Editors, with one exception ; some were whole time or pure editors, others part time or unpure editors. The former usually practised the Lancashire precept, " Think owt, say nowt."

Dr. MacNalty, in reply, described the origin of the Joint Council, its place in the national antituberculosis campaign, and his own association with the earlier years of the Council.

Sir Henry Gauvain, in proposing the health of Council, related a series of interesting and whimsical experiences which befell him while participating in the British Medical Association World Tour. In reply to this toast Dr. Ernest Ward mentioned the impression made upon him by the remark of a leading Medical Officer of Health that tuberculosis work must be regarded as static. We should try to belie this conception. The mortality rate steadily diminished, but in Italy, he was told, the diminution was swifter. The increase in collapse therapy had made no visible impression in the tuberculosis death-rate. Was this because the time was too short, or was there some other reason ? Ninety per cent. of cases of tuberculosis were infected from an unknown source ; until these sources could be traced our work was handicapped. He believed more radiography of the adults in the house where a case of tuberculosis had occurred would be helpful. Dr. Hawthorne thanked our host and proposed his health in a witty and apposite speech, to which Dr. Cox replied, and thus ended an eventful and successful party.

TUBERCULOSIS ASSOCIATION

At a meeting of the Association held at Manson House on Friday, January 17, Dr. Noel Bardswell opened a discussion on After-Care of the Tuberculous in London. He stressed the importance of housing and stated that the London County Council have made an enormous improvement in the housing conditions of the poor. Every effort is made to keep children away from infection, and on an average 150 children are away from home for this purpose at any one time. Dr. Bardswell referred to arrangements for giving artificial pneumothorax refills, and said that these, like tuberculin injections, had the additional advantage of getting the patient to attend the clinic regularly and so keep under supervision.

Mr. J. G. Johnston discussed the problem as regards surgical tuberculosis chiefly in children up to 16 years of age. Mortality from this type of the disease is small, but it is most important to avoid deformities, and many children are being supplied with splints, which are renewed when necessary.

Miss Marx said that the London method of dealing with this problem was

ahead of those of other countries. She regarded the term " after-care " as mistaken, because care of the home should also be taken during the illness of the patient. If the patient was the bread-winner, it was essential to save him as far as possible from anxiety as to conditions at home. Occupation, extra nourishment and help in other ways was often necessary for other members of the family.

When the patient left a sanatorium it was important to see that he did not infect others at home. Hostels for T.B. positive cases or colonies might provide for a few such cases, but probably less than 10 per cent. of them were suitable for colony life.

At the evening session Dr. James Maxwell read a paper on Intestinal Tuberculosis. His work was based on 8,087 autopsies, in which signs of active tuberculosis were found in 785. Of these, intestinal tuberculosis was present in 233. He suggested that the terms primary and secondary should be discarded and advocated the following classification :

Nodular ulcerative :

(*a*) Simple.
(*b*) With pulmonary tuberculosis.
(*c*) With miliary tuberculosis.
Hyperplastic—*e.g.*, cæcum.
Lymphatic.
Adhesive.
Extrinsic, or where the disease was an extension from elsewhere—*e.g.*, the hip.

He thought that the frequency of tuberculosis in the ileum and cæcum was due to the excess of lymphoid tissue in those regions. In the stomach, where there is little lymphoid tissue, tuberculosis is rare.

MEETINGS were held at Manson House on February 21, 1936. Dr. H. H. Scott opened a discussion on " Primary Tuberculosis in Children and its Relationship to Meningitis."

Dr. Scott reviewed the old theory that tuberculous meningitis was usually part of a general tuberculosis, the primary seat being often in the bronchial, cervical or mesenteric glands, by the infective material passing to the meninges by way of the lymph or blood stream. He argued that as so many cases, where the primary focus was in the lungs, showed no tuberculosis of any secondary site except the meninges, it was difficult to believe in blood infection, although he knew of no lymphatic connection between the lungs and the base of the brain. Tuberculous meningitis might occasionally be primary, as he certainly had one definite case in a child 4 years old. In

his series of autopsies on tuberculous subjects, meningeal infection was found in 41 per cent. of 225 children under 10 years, and in 49 per cent. of 65 adults over 20 years.

He criticised the statement that tubercles may often be found along the fissures of Sylvius when they are not discoverable elsewhere in the meninges. Tuberculous tumours of the brain or conglomerate tubercle might or might not be associated with meningitis. They were often multiple, and had a predilection for the cerebellum, the base of the brain, and the basal ganglia.

Dr. C. H. C. Toussaint dealt with relationship between bovine infection and tuberculous meningitis. He summarised recent work and showed that 25 per cent. of cases were due to bovine infection, and 75 per cent. due to the human type, thereby stressing the importance of human infection in childhood. He then dealt with a series of 80 children. These were contact cases which died of tuberculosis in Bermondsey between 1928 and 1935. In 46 out of 54 cases the probably human source of infection could be traced.

At the evening session Sir Henry Gauvain read a paper on " The Treatment of Tuberculous Lesions of Bones and Joints."

He stressed the importance of remembering that a tuberculous bone or joint lesion was secondary to a primary focus occurring elsewhere.

Treatment therefore followed on two lines, local and general. Extirpation of the lesion became less often indicated, although it was still of value in certain cases, such as tuberculous disease of the knee-joint in an adult. In conservative treatment, three main lines must be followed—to improve the patient's general health, to increase his powers of resistance to tuberculous disease, and to preserve or restore the part attacked.

He discussed the relative value of bone grafting or fusing and mechanical immobilisation in various types of lesions, and favoured conservative treatment in the majority of cases, although in adults with tuberculous knee-joints he usually favoured excision. He gave illustrations of the value of sun bathing, light treatment, and sea bathing and showed an interesting series of slides of cases in which treatment had had beneficial results.

Mr. G. R. Girdlestone followed with a detailed analysis of nearly 300 cases, and whilst he favoured conservative treatment in children, he considered that the problem of adult cases demanded other measures. He thought that the factors which decided operative intervention were : the preservation of life, the elimination of persistent disease, and the aim of providing a permanently safe and useful limb.

The economic factor had also to be considered in adults.

By a series of slides he showed the proportion of cases treated by various methods and the results obtained.

REVIEWS OF NEW BOOKS

The Tuberculin Handbook. By HALLIDAY SUTHERLAND, M.D. Pp. 96. 1936. Price 7s. 6d. Oxford University Press.

The author states in the preface of this book that tuberculin has been the subject of controversy for over forty years. When the discovery of the tubercle bacillus by Robert Koch was followed by his production of tuberculin, hopes that it would prove to be a specific cure ran high. It was found, however, that the value of tuberculin was not like that of a very few other later discoveries such as insulin or anti-diphtheritic serum, and it began to fall into disuse. This, no doubt, was largely due to the undue enthusiasm which accompanied its discovery. It was, and still is, regarded by many not only as a failure but as being actually dangerous. Dr. Sutherland says, " Tuberculin will not resuscitate the dying "; but this does not prove that it is useless. Large doses and careless administration are dangerous, but the same applies to morphia and most drugs. We think that there is too great a tendency to take it for granted that tuberculin has been weighed in the balance and found wanting.

After a brief description of the history of the discovery of tuberculin and of the different types which are on the market, the author devotes a chapter to the early signs of pulmonary tuberculosis. In the next chapters a detailed description of the different cutaneous tests is given, and the information which may be derived from their results is discussed.

The subcutaneous test is next considered, and the author holds the view that by this means an old arrested lesion can be differentiated from an active one, the cutaneous tests being regarded as showing evidence of infection and the subcutaneous one evidence of activity and also as an indication of dosage should treatment by tuberculin follow.

Finally the method of treatment is described, dosage, intervals between injections and treatment of febrile and other reactions being fully considered.

Details which are not easily found in other works are given in this little volume. For example, the comparative strengths of the various tuberculins, methods of dilution and the deterioration of different dilutions if kept, clear instructions as to dosage, not only in treatment but in testing, are all given.

Those few remaining disciples who regard tuberculin as specific and the only real treatment for tuberculosis may think that Dr. Sutherland has not gone far enough, but those who take the extreme opposite view cannot accuse him of going too far, for the author makes no extravagant claims, and the book gives a fair and clear account of the possibilities of tuberculin for diagnosis and treatment, and can be warmly recommended to all who are interested in the problem of tuberculosis.

Clinical Tuberculosis. Edited by Dr. Benjamin Goldberg, M.D., F.A.C.P., Associate Professor of Medicine, University of Illinois College of Medicine. Philadelphia: F. A. Davis and Co., 1935. In two volumes.

This is a treatise on tuberculosis by many authors and will make a valuable addition to the library of anyone interested in this disease. It deals with tuberculosis in all forms, and every aspect is given full consideration. The first chapter on epidemiology by Dr. Drolet contains information and statistics relating to the effect of age, sex, race, housing and other social conditions on the prevalence and mortality of tuberculosis. The author emphasises the fact that although the mortality amongst the negroes in the United States is much higher than that amongst the white population, the percentage of positive reactors to tuberculin in children is about the same, whatever their race may be. The varieties of tubercle bacilli, the isolation of them from the blood, and unusual manifestations of infection, such as Boek's sarcoid, are all described. Modern views on allergy, reaction and immunity are discussed in an interesting chapter by Drs. Koch and Mellor, and Dr. Jaffé contributes the chapter on patiology.

Perhaps one of the most interesting chapters is that on classification by Drs. Ornstein and Ulmer. Much information which is not contained in the ordinary textbook will be found here, and as examples we may mention the twelve pages by Dr. Goldberg devoted to diet, and the chapter by Dr. Neyman dealing with the psychology of tuberculosis.

Section D, by Drs. R. C. and R. W. Watson, in the first volume consists of 225 pages dealing with collapse therapy, and gives a very clear account of the modern methods of this treatment. Some may think that more space should have been given to this most important subject, but at least the authors are to be congratulated on not having padded their article by unnecessary details and numerous illustrations of the different forms of instruments and various technique. The essentials are there.

Tuberculin was hailed as a cure for tuberculosis, but proved so disappointing that it fell into disuse, though later it became a recognised method of testing for infection. Dr. Poncher gives a very fair summary of the present value and limitations of tuberculin, and discusses fully the errors in the clinical interpretation of the tuberculin reaction.

One chapter describes bronchoscopy in tuberculosis, and Dr. Clerf discusses this method of investigation in non-tuberculous conditions, although its value in tuberculosis has not yet been proved.

Dr. Lewy contributes a chapter on trauma and tuberculosis for industrial and compensatory consideration, and this is full of information and suggestions. The lesson of the Great War was that injury played little, if any, part in activating and none in originating tuberculosis, although, if the disease developed after injury, the hearts of the jury often take possession of their heads in finding a verdict. When one remembers that out of 824 persons in England and Wales who died of tuberculosis in 1932 it was the pulmonary form in 690, one can appreciate the necessity of devoting the majority of space to pulmonary tuberculosis in such a work. Tuberculosis of other organs has not, however, been neglected, and there are articles dealing with tuberculosis of the bones, joints, genito-urinary system, eye,

skin, abdomen, ear, nose and throat, and, indeed, all the manifestations of tuberculosis find a place in this work. The practitioner in difficulties on some point connected with tuberculosis will get valuable information upon almost any aspect of the disease. The specialist will regard it as a standard work, more valuable, perhaps, because the authors give their own experience and views and limit their references to other authors. References there are, but we congratulate the editor in avoiding the common mistake of including a large number of references to brief articles in foreign languages, which are as unimportant as they are difficult to obtain.

The illustrations, charts and X-rays are good and well produced, and here again the authors were wise to avoid excess.

Milk Production and Control. By W. C. HARVEY, M.D., Medical Officer of Health, Borough of Southgate; and H. HILL, M.R.S.I., Sanitary Inspector, Borough of Southgate. London: H. K. Lewis and Co., 1936. Pp. 555. Price 21s.

The recent discussions about milk supply make the publication of this book especially welcome.

The authors describe the chemical composition of milk and emphasise its value as a food. One quart of milk equals in food value 1 lb. of lean meat or 2 lb. of potatoes. One glassful is equal to two eggs. For school children it may be regarded as almost an essential article of diet, and it is therefore of the greatest importance to ensure a cheap and sufficient supply of safe milk. Tuberculosis and other diseases lie hidden in milk, and it is stated that approximately 6 per cent. of all deaths from tuberculosis are of bovine origin, and some 2,000 deaths occur annually in this country from bovine tuberculosis.

The effect of heat on milk is fully discussed and methods of pasteurisation described in detail.

The ordinary layman will find much of interest in this book, and the expert will find much valuable information.

Details of laboratory methods of milk examination, the transport of milk and how to take care of it in the home are fully described. The yields of milk that may be expected from different breeds of cattle and the butter-fat percentage are all given.

Two chapters of special interest deal with the cowshed and care of the cows, and make one realise how much modern improvements have done in this direction, not only to ensure the cleanliness of the milk, but for the comfort of the cattle.

One chapter deals with legislation, and the next with the future of the milk industry.

The book, which concludes with an appendix, a useful list of references, and a full index, is one of great value and interest, and can be warmly recommended.

The Diagnosis and Treatment of Pulmonary Tuberculosis. By JOHN B. HAWES, M.D., President of the Boston Tuberculosis Association; and MOSES J. STONE, M.D., Assistant Professor Diseases of the Chest, Boston University School of Medicine. London: Henry Kimpton, 1936. Pp. 215. Price 12s. 6d.

This little book is described as a handbook for practitioners and a textbook for students, nurses and social workers. The authors are to be congratulated on getting so much information into such small space. Nothing has been forgotten, although space prevents more than the briefest mention of subjects about which volumes have been written elsewhere. Thus only half a page is devoted to tuberculin and still smaller space to sanocrysin.

The first nine chapters deal with diagnosis, physical signs and methods of examination, and the next eight with treatment. One chapter devoted to diet is brief but noteworthy.

The final chapters deal with tuberculosis in old age, in relationship to marriage and pregnancy, dangerous trades and prevention.

The book is a useful summary of modern methods of diagnosis and treatment of pulmonary tuberculosis.

An Index of Treatment. Edited by Robert Hutchinson, M.D., LL.D., F.R.C.P. Eleventh edition. Pp. xvi+1020, with 147 illustrations. Price £2 2s. John Wright and Sons, Ltd.

One cannot but wholeheartedly welcome the eleventh edition of such a classic work. It must be very seldom that such a great deal of talent, in the form of specialists in their own subject, has been gathered together in one volume and put at the disposal of others. The Editor's remarks on the principle of Therapeutics are masterly.

There are many new articles dealing with subjects not mentioned in previous editions; to quote but one or two—Dr. Bray on Allergic Diseases, about which he has made an extensive contribution to the literature and which he has placed on a rational basis; and Professor Ellis on Alkalosis, a subject that has come into prominence much more since the introduction of various patent alkali powders to the general public.

Many sections of the work have been completely revised and rewritten in the light of modern medical knowledge, which, owing to its rapid advancement, necessitates many more editions—at shorter intervals—of well-known books than was previously the case. The article by Dr. Burrell on Pulmonary Tuberculosis, considering that such a complex disease has to be dealt with in such a short space, is of the highest order and could be read by both General Practitioner and Specialist with great advantage. Mr. Tudor Edwards and Dr. Chandler have also contributed extremely good articles on chest problems. The article on Pulmonary Abscess clearly shows the danger of Artificial Pneumothorax as a mode of treatment and leaves no doubt in the mind that it should be employed only in the rarest of cases of this nature.

It is difficult to single out articles for praise in a book of this type, as all are worthy of it. In conclusion, all who are intimately concerned with Therapeutics, especially the busy general practitioner, will find it an invaluable aid in daily practice.